

WORK and the HEART

Transactions of the First Wisconsin Conference on Work and the Heart

Held in Milwaukee and sponsored jointly by the Wisconsin Heart Association and the Marquette University School of Medicine in co-operation with the American Heart Association, the National Heart Institute, and the Industrial Health Council of the American Medical Association

Editors

FRANCIS F. ROSENBAUM, M D

ELSTON L. BELKNAP, M D

Associate Editors

RODNEY R. BEARD, M D

HOWARD B. BURCHELL, M D, P H D

HOWARD L. CORRELL, M D

CHARLES W. CRUMPTON, M D

JESSE E. EDWARDS, M D

BARNETT S. FOX

LEONARD J. GOLDWATER, M D

GEORGE A. HELLMUTH, M D

EDWARD M. KLINE, M D

CHESTER M. KURTZ, M D

JOSEPH F. KUZMA, M D

PHILIP R. LEE, M D

LAMONT R. SCHWEIGER, M D

JAMES J. SMITH, M D, P H D

DAVID M. SPAIN, M D

DONAL R. SPARKMAN, M D

WILLIAM H. STEWART, M D

MAURICE B. VISSCHER, M D, P H D

WORK

TRANSACTIONS OF THE FIRST

Edited by

FRANCIS F. ROSENBAUM, M.D.

*Associate Clinical Professor of Medicine
Marquette University School of Medicine*

and

ELSTON L. BELKNAP, M.D.

*Professor and Director
Department of Occupational and Environmental Medicine
Marquette University School of Medicine*

and the HEART

WISCONSIN CONFERENCE ON WORK AND THE HEART



PAUL B. HOEBER, INC.

Medical Book Department of Harper & Brothers

WORK AND THE HEART

TRANSACTIONS OF THE FIRST WISCONSIN CONFERENCE
ON WORK AND THE HEART

*Copyright © 1959, by Paul B. Horber, Inc.,
Medical Book Department of Harper & Brothers
Printed in the United States of America*

*All rights reserved
For information address Paul B. Horber, Inc.,
Medical Book Department of Harper & Brothers,
19 East 33rd Street, New York 16, N.Y.*

x-i

Library of Congress catalog card number 59-11092

CONTENTS

	ACTIVE CONFERENCE PARTICIPANTS	xj
	PREFACE	
	<i>Francis F Rosenbaum</i>	xvii
	INTRODUCTION	
	<i>E Coules Andrus</i>	xx
PART I	BASIC PHYSIOLOGY	
CHAPTER 1.	THERMODYNAMICS OF MUSCLE	
	<i>Robert W Ramsey</i>	3
CHAPTER 2.	MOVEMENTS AND CONTRACTILE PROTEINS OF MUSCLES AND CELLS	
	<i>Hans H Weber</i>	8
CHAPTER 3.	HEMODYNAMIC DETERMINANTS OF THE OXYGEN CONSUMPTION OF THE HEART WITH SPECIAL REFERENCE TO THE TENSION-TIME INDEX	
	<i>S J Sarnoff, E Braunwald, G. H. Welch, W. N. Stainsby, R. B. Case, and R. Macruz</i>	18
CHAPTER 4.	THE HEMODYNAMIC EFFECTS OF QUANTITATIVE AND CONTROLLED MITRAL AND AORTIC VALVULAR REGURGITATION: PHYSIOLOGIC AND CLINICAL CONSIDERATIONS	
	<i>E Braunwald, G. H. Welch, and S. J. Sarnoff</i>	34
CHAPTER 5.	CORONARY BLOOD FLOW AND CARDIAC ENERGETICS	
	<i>Steven M Horvath</i>	48

CHAPTER 6.	CONTROL OF CARDIAC OUTPUT IN MAN <i>James V' Warren</i>	54
CHAPTER 7.	REGULATION OF THE HEART RATE <i>D. B. Dill</i>	60
CHAPTER 8.	THE IMPORTANCE OF THE SIZE AND SHAPE OF THE HEART <i>A C Burton</i>	74
CHAPTER 9.	CARDIAC FUNCTION IN CONGESTIVE HEART FAILURE IN MAN <i>Ludwig W' Eichna</i>	76
CHAPTER 10.	ENERGETICS OF THE HEART AND CORONARY FLOW <i>Louis N Katz</i>	86
	REPORT OF PANEL ON BASIC PHYSIOLOGY <i>William H Stewart</i>	98
PART II CLINICAL PHYSIOLOGY		
CHAPTER 11.	OCCUPATIONAL AND ENVIRONMENTAL FACTORS IN THE DEVELOPMENT OF HEART DISEASE <i>Ansel Keys</i>	103
CHAPTER 12.	RELATIONSHIP OF PHYSICAL ACTIVITY TO SERUM CHOLESTEROL CONCENTRATION <i>Henry Longstreet Taylor</i>	111
CHAPTER 13.	HEART DISEASE IN INDIANA FARMERS <i>W' H M Morris</i>	117
CHAPTER 14.	ENERGY EXPENDITURE BY CARDIAC AND NONCARDIAC FACTORY WORKERS <i>Herman K Hellerstein and Amasa B Ford</i>	122
CHAPTER 15.	THE MEASUREMENT OF CARDIAC PERFORMANCE IN OSTENSIBLY WELL, OLDER INDIVIDUALS <i>Milton Landowne</i>	132
CHAPTER 16.	EVALUATION OF FUNCTIONAL CAPACITY IN PATIENTS WITH CARDIOVASCULAR DISEASE <i>Robert A. Bruce, Donal R Sparkman, Robert M. Levenson, and Albert M Hurley</i>	140
CHAPTER 17.	INFLUENCE OF HOT AND HUMID ENVIRONMENT UPON THE WORK OF THE HEART <i>George E. Burch</i>	148
CHAPTER 18.	RELATIVE BURDENS OF EMOTION AND EXERCISE UPON THE CARDIOVASCULAR SYSTEM <i>Stewart Wolf</i>	158

CHAPTER 19.	FACTORS RELATED TO PHYSICAL UNFITNESS IN NEUROCIRCULATORY ASTHENIA <i>Gunnar Björck</i>	170
CHAPTER 20.	LABORATORY TESTS FOR PHYSICAL FITNESS <i>Gunnar Björck</i>	177
CHAPTER 21.	EFFECT OF WORK ON THE HEART <i>Lucien A Brouha</i>	180
CHAPTER 22.	THE LAURU FORCE PLATFORM <i>Lucien A Brouha</i>	196
CHAPTER 23.	EFFECTS OF VIGOROUS EXERCISE ON THE HEART <i>Martti J Karjonen</i>	199
CHAPTER 24.	WORK AND ENVIRONMENTAL STRESS IN THE CARDIAC FARMER <i>W H M Morris</i>	211
CHAPTER 25.	THE METABOLIC AND CARDIAC DEMANDS OF HOSPITAL AND REHABILITATION ACTIVITIES <i>William G Kubicek, Frederic J Kottke, and Jean N Danz</i>	215
	REPORT OF PANEL ON CLINICAL PHYSIOLOGY <i>Howard B Burchell</i>	224
PART III	PATHOLOGY	
CHAPTER 26.	INTRAMURAL HEMORRHAGES AND THE HISTOGENESIS OF ATHEROSCLEROSIS <i>John D Hamilton and J H Mowbray</i>	233
CHAPTER 27.	THE CAUSAL RELATIONSHIP OF STRESS TO INTIMAL HEMORRHAGE AND CORONARY OCCLUSION <i>J C Paterson</i>	243
CHAPTER 28.	THE PATHOLOGY OF THE CONDUCTION SYSTEM AS RELATED TO GENERAL CARDIAC PATHOLOGY <i>Maurice Lev</i>	251
CHAPTER 29.	THE STIMULUS TO REVASCULARIZATION OF MUSCLE AN EXPERIMENTAL INQUIRY <i>George M Hass, Richard H Andresen, Clarence W Monroe, Fay Squire, and Dorothy A Madden</i>	257
CHAPTER 30.	INCIDENCE OF UNSUSPECTED HEALED MYOCARDIAL INFARCTION IN A GENERAL HOSPITAL <i>S E Gould and L P Cawley</i>	269

APTER 31.	ANALYSIS OF THE INCREASE IN MYOCARDIAL INFARCTIONS IN MALMO, SWEDEN, 1935-1954 <i>Gunnar Björck</i>	274
APTER 32.	RELATION OF SEX, AGE, AND PHYSICAL ACTIVITY TO SUD- DEN DEATH FROM CORONARY ARTERIAL OCCLUSION <i>David M Spain and Victoria A Bradess</i>	283
APTER 33.	CIRCUMSTANCES RELATED TO SUDDEN, UNEXPECTED DEATH IN CORONARY HEART DISEASE <i>Sidney B Weinberg and Milton Helpern</i>	288
APTER 34.	THE RELATIVE ETIOLOGICAL IMPORTANCE OF VARIOUS STRESSES IN CARDIAC DISORDERS <i>William Dock</i>	293
	REPORT OF THE PANEL ON PATHOLOGY <i>Jesse E Edwards</i>	296
PART IV	WORK CLASSIFICATION	
APTER 35.	EXPERIENCE OF THE WORK CLASSIFICATION UNIT AT BELLEVUE HOSPITAL <i>Lewis H Bronstein</i>	305
APTER 36.	EXPERIENCE OF THE CARDIAC WORK CLASSIFICATION UNIT IN BOSTON, MASSACHUSETTS <i>Richard J Clark</i>	311
APTER 37.	EXPERIENCE AT THE CARDIAC WORK CLASSIFICATION UNIT OF THE HEART ASSOCIATION OF SOUTHEASTERN PENNSYL- VANIA <i>David Gelfand</i>	322
APTER 38.	RESULTS OF STUDIES AT THE WORK CLASSIFICATION CLINIC OF THE CLEVELAND AREA HEART SOCIETY <i>T. V' Parran, H. D Hellerstein, D Cohen, and E Goldston</i>	330
APTER 39.	WORK CLASSIFICATION UNIT SURVEY <i>Sidney S Sobin</i>	340
APTER 40.	EXPERIENCE WITH CARDIACS AT THE ALTRO WORKSHOPS <i>Abraham Jezer, B J Black, Daniel Adelson, Florence Haselkorn, Leopold Bellak, Gloria Auerhan, Irwin Friedman, Hannah Chrystall, Annabelle Re- plansky, Jr, Bruria Szapira, Marian F Loew, Shirley Madonick, and Libby Eigen</i>	348

CHAPTER 41.	THE SELECTIVE PLACEMENT OF CARDIACS IN INDUSTRY <i>Neill K. Weaver</i>	368
CHAPTER 42.	A STUDY OF EMPLOYEES WHO DEVELOPED MYOCARDIAL INFARCTION <i>C A D'Alonzo and Sidney Pell</i>	375
CHAPTER 43.	EXPERIENCE IN SELECTIVE PLACEMENT AND FOLLOW-UP ON CARDIACS <i>William E R Greer</i>	383
CHAPTER 44.	MYOCARDIAL INFARCTION IN GARMENT WORKERS <i>Leo Price</i>	387
CHAPTER 45.	EMPLOYMENT PROBLEMS IN PERSONS WITH CARDIOVASCULAR DISEASE <i>E A Irwin</i>	395
CHAPTER 46.	HEART DISEASE AND FITNESS FOR WORK <i>A Morgan Jones</i>	400
	REPORT OF PANEL ON WORK CLASSIFICATION <i>Leonard Goldwater</i>	410
PART V WORKMEN'S COMPENSATION		
CHAPTER 47.	MAGNITUDE OF THE PROBLEM OF THE CARDIAC-IN-INDUSTRY <i>Harry E Ungerleider and Richard S Gubner</i>	417
CHAPTER 48.	CAUSAL RELATIONSHIPS IN HEART DISEASE IN WORKMEN'S COMPENSATION CASES <i>Meyer Texon</i>	426
CHAPTER 49.	EMPLOYMENT OF THE CARDIAC <i>Irvn Klein</i>	432
CHAPTER 50.	WORKMEN'S COMPENSATION—AN INDUSTRIAL AND SOCIAL MECHANISM <i>Henry D Sayer</i>	438
CHAPTER 51.	CAN WE REHABILITATE WORKMEN'S COMPENSATION? <i>Kenneth E Pohlmann</i>	454
CHAPTER 52.	WORKMEN'S COMPENSATION AND ITS RELATIONSHIP TO THE HEART <i>Theodore C Waters</i>	460
CHAPTER 53.	MEDICAL WITNESSES IN WORKMEN'S COMPENSATION <i>Ivan C. Rutledge</i>	467

CHAPTER 54.	RELATIONSHIP OF EMPLOYMENT TO THE DEVELOPMENT OF CARDIAC PATHOLOGY	
	<i>R G Knutson</i>	480
CHAPTER 55.	THE UTAH MEDICAL PANEL SYSTEM	
	<i>Otto A Wiesley</i>	482
CHAPTER 56.	THE UTAH PLAN FOR EVALUATING INDUSTRIAL CARDIAC CASES	
	<i>Louis E Viko</i>	487
CHAPTER 57.	DISABILITY DUE TO CARDIAC IMPAIRMENT UNDER THE DIS- ABILITY INSURANCE PROGRAM	
	<i>Arthur B Price</i>	499
	REPORT OF PANEL ON WORKMEN'S COMPENSATION	
	<i>Rodney R Beard</i>	507
PART VI	OUTCOME AND PROSPECTS	
	CONFERENCE SUMMARY	
	<i>Maurice Visscher</i>	517
	EPILOGUE	
	<i>Elston L Belknap</i>	521
	INDEX	523

ACTIVE CONFERENCE PARTICIPANTS

- E. Cowles Andrus, M.D.**, Physician-in-Charge, Adult Cardiac Clinic, Johns Hopkins Hospital; Associate Professor of Medicine, Johns Hopkins University School of Medicine, Baltimore, Senior Scientific Advisor, National Heart Institute, Bethesda, Maryland
- Rodney R. Beard, M.D., M.P.H.**, Professor of Preventive Medicine, Stanford University School of Medicine, San Francisco; Clinical Professor of Occupational Health, University of California School of Public Health, Berkeley, California
- Elston L. Belknap, M.D.**, Professor and Director of Department of Occupational and Environmental Medicine, Marquette University School of Medicine, Medical Director, Globe-Union Incorporated, Milwaukee, Wisconsin
- Gunnar Biorck, M.D.**, Associate Professor of Internal Medicine, University of Lund, Cardiological Laboratory, Malmö, Sweden *Now* Professor of Medicine, Karolinska Institute, Stockholm, Sweden
- Eugene Braunwald, M.D.**, Chief, Section of Cardiology, Clinic of Surgery, National Heart Institute, Bethesda; Assistant in Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland
- Lewis H. Bronstein, M.D.**, Director, Work Classification Unit, and Associate Medical Director, Beekman Downtown Hospital, New York, New York
- Lucien Brouha, M.D., D.Sc.**, Chief Physiologist, Haskell Laboratory for Toxicology and Industrial Medicine, Newark, Delaware, Honorary Professor, University of Liege, Belgium
- Robert A. Bruce, M.D.**, Associate Professor of Medicine in Cardiology, University of Washington School of Medicine, Seattle, Washington

- George Burch, M.D., Henderson Professor of Medicine, Tulane University School of Medicine, Physician in Chief, Tulane Unit, Charity Hospital, New Orleans, Louisiana
- Howard B. Burchell, M.D., Ph.D., Consultant in Medicine, Mayo Clinic; Professor in Medicine, *The Mayo Foundation for Medical Education and Research*, University of Minnesota Graduate School, Rochester, Minnesota
- Alan C. Burton, Ph.D., Professor of Biophysics, University of Western Ontario Faculty of Medicine, London, Ontario, Canada
- Richard J. Clark, M.D., Director, Cardiac Work Classification Unit, Massachusetts Heart Association, Bay State Medical Rehabilitation Clinic; Associate Physician, Massachusetts General Hospital, Boston, Massachusetts
- Howard L. Correll, M.D., Co-ordinator of Cardiovascular Teaching and Research and Associate Clinical Professor of Medicine, Marquette University School of Medicine, Chief of Staff, St. Luke's Hospital, Milwaukee, Wisconsin
- Charles W. Crumpton, M.D., Director, Cardiovascular Research Laboratory, and Associate Professor of Medicine, University of Wisconsin School of Medicine, Madison, Wisconsin
- C. Anthony D'Alonzo, M.D., F.A.C.P., Assistant Medical Director, E. I. du Pont de Nemours and Company, Inc., Chief of Industrial Medicine, Memorial Hospital, Wilmington, Delaware
- D. B. Dill, Ph.D., Deputy Director of Medical Research, U.S. Army Chemical Warfare Laboratories, Army Chemical Center, Maryland, Visiting Lecturer in Physiology, Harvard School of Public Health, Boston, Massachusetts
- William Dock, M.D., Former Cardiologist, Palo Alto Medical Clinic, Palo Alto, California; Professor of Medicine, State University of New York, Downstate Medical Center, Brooklyn, New York
- Jesse E. Edwards, M.D., Consultant, Section of Pathologic Anatomy, Mayo Clinic; Professor of Pathologic Anatomy, *The Mayo Foundation for Medical Education and Research*, University of Minnesota Graduate School, Rochester, Minnesota
- Ludwig W. Eichna, M.D., Associate Professor of Medicine, New York University College of Medicine, Visiting Physician, Bellevue Hospital, New York, New York
- Amasa B. Ford, M.D., Senior Instructor in Medicine, Western Reserve University School of Medicine, Assistant Physician, University Hospitals, Cleveland, Ohio
- Barnett S. Fox, Executive Secretary, American Academy of Compensation Medicine; Lecturer on Industrial Medicine, New York University Post-Graduate Medical School, New York, New York

- David Gelfand, M.D., F.A.C.P., F.C.C.P., F.A.C.C., Director, Cardiac Work Classification Unit, Heart Association of Southeastern Pennsylvania, Assistant Professor of Cardiology, University of Pennsylvania Graduate School of Medicine, Cardiologist, Philadelphia General Hospital and Kensington-Maimonides Hospital, Philadelphia, Pennsylvania
- Leonard J. Goldwater, M.D., D.Sc.Med., Consultant in Social and Occupational Health, World Health Organization, Professor of Occupational Medicine, Columbia University School of Public Health and Administrative Medicine, New York, New York
- S. E. Gould, M.D., D.Sc., Professor of Pathology, Wayne State University College of Medicine, Detroit, Director of Laboratories, Wayne County General Hospital, Eloise, Michigan
- William E. R. Greer, M.D., Medical Director, Gillette Safety Razor Company; Assistant Professor of Industrial Medicine, Boston University School of Medicine, Boston, Massachusetts
- John D. Hamilton, M.D., Professor and Head of Department of Pathology, University of Toronto Faculty of Medicine, Pathologist-in-Chief, Toronto General Hospital, Toronto, Canada
- George M. Hass, M.D., Chairman, Department of Pathology, Presbyterian Hospital, Professor of Pathology, University of Illinois College of Medicine, Chicago, Illinois
- Herman K. Hellerstein, M.D., Assistant Professor of Medicine, Western Reserve University School of Medicine, Director, Work Classification Clinic, Cleveland Area Heart Society, Cleveland, Ohio
- George A. Hellmuth, M.D., Director, Cardiovascular Section, Marquette University School of Medicine, Chief of Cardiology, Milwaukee County Hospital, Milwaukee, Wisconsin
- Milton Helpert, M.D., Chief Medical Examiner, City of New York, Professor of Forensic Medicine, New York University Post-Graduate Medical School, New York, New York
- Steven M. Horvath, Ph.D., Director, Institute of Gerontology, and Professor of Physiology, State University of Iowa College of Medicine, Iowa City, Iowa. Now Adjunct Professor, Applied Physiology, University of Pennsylvania School of Medicine. Head of Department of Physiology, Division of Research, Lankenau Hospital, Philadelphia, Pennsylvania
- Earle A. Irvin, M.D., Chairman, Rehabilitation Committee, American Heart Association, Medical Director, Ford Motor Company, Dearborn, Michigan
- Abraham Jezer, M.D., Medical Director, Cardiac Project, Altro Workshops; Attending Physician, Montefiore Hospital, New York, New York
- A. Morgan Jones, M.B., F.R.C.P., Director, Department of Cardiology, University of Manchester, Manchester, England

- Martti J. Karvonen, M.D., Ph.D.,** Director, Physiological Department, Institute of Occupational Health, Helsinki, Finland
- Louis N. Katz, M.D.,** Director, Cardiovascular Department, Medical Research Institute, Michael Reese Hospital; Professorial Lecturer, Department of Physiology, University of Chicago School of Medicine, Chicago, Illinois
- Ancel Keys, Ph.D.,** Professor and Director, Laboratory of Physiological Hygiene, University of Minnesota, Minneapolis, Minnesota
- Irvin Klein, M.D.,** Associate Compensation Examining Physician, Workmen's Compensation Board, New York State; Member, Work Classification Unit, Cardiac Clinic, Beekman Hospital, New York, New York
- Edward M. Kline, M.D.,** Consulting Physician, Lamp Division, General Electric Company; Senior Clinical Instructor of Medicine, Western Reserve University School of Medicine, Cleveland, Ohio
- R. G. Knutson,** Chairman, The State of Wisconsin Industrial Commission, Madison, Wisconsin
- W. G. Kubicek, Ph.D.,** Professor of Physical Medicine and Rehabilitation, University of Minnesota School of Medicine, Minneapolis, Minnesota
- Chester M. Kurtz, M.D.,** Associate Clinical Professor of Medicine, University of Wisconsin School of Medicine, Madison, Wisconsin *Now* Manager, Veterans Administration Hospital, Fort Bayard, New Mexico
- Joseph F. Kuzma, M.D.,** Director, Department of Pathology, Marquette University School of Medicine, Milwaukee, Wisconsin
- Milton Landowne, M.D.,** Medical Director, Levindale Hebrew Home and Infirmary; Chief, Division of Chronic Diseases, Sinai Hospital; Assistant Professor of Medicine, The Johns Hopkins University School of Medicine, Baltimore, Maryland
- Philip R. Lee, M.D.,** Internist, Palo Alto Medical Clinic, Palo Alto; Clinical Instructor in Medicine, Stanford University School of Medicine, San Francisco, California
- Maurice Lev, M.D.,** Director, Congenital Heart Disease Research and Training Center, Hektoen Institute, Professor of Pathology, Northwestern University Medical School, Chicago, Illinois
- Alan R. Moritz, M.D.,** Chairman, Committee on Legal Medicine, American Medical Association, Professor of Pathology, Western Reserve University School of Medicine, Cleveland, Ohio
- W. H. M. Morris, Ph.D.,** Assistant Professor of Agricultural Economics, Purdue University, Lafayette, Indiana
- Theodore V. Parran, M.D.,** Associate Director, Work Classification Clinic, Cleveland Area Heart Society; Medical Consultant, Cleveland Vocational Guidance and Rehabilitation Services, Cleveland, Ohio

- J. C. Paterson, M.D., Professor of Medical Research, University of Western Ontario Faculty of Medicine; Pathologist, Westminster Hospital, Department of Veterans' Affairs, London, Ontario, Canada
- Kenneth E. Pohlmann, A.B., A.M., Rehabilitation Officer, United Mine Workers of America, Welfare and Retirement Fund, Washington, D C
- Arthur B. Price, M.D., M.P.H., Chief Medical Consultant, Bureau of Old-Age and Survivors Insurance, Department of Health, Education, and Welfare, Baltimore, Maryland. *Now* Regional Medical Director, Public Health Service, Department of Health, Education, and Welfare, Chicago, Illinois
- Leo Price, M.D., Director, Union Health Center, Associate Clinical Professor of Industrial Medicine, New York University College of Medicine, New York, New York
- Robert W. Ramsey, Ph.D., Professor of Physiology, Medical College of Virginia, Richmond, Virginia
- Francis F. Rosenbaum, M.D., Associate Clinical Professor of Medicine, Marquette University School of Medicine; Chief, Cardiac Clinic, Milwaukee Children's Hospital, Milwaukee, Wisconsin
- Ivan C. Rutledge, LL.B., LL.M., Professor of Law, Indiana University School of Law, Bloomington, Indiana
- Stanley J. Sarnoff, M.D., Director, Laboratory of Cardiovascular Physiology, National Heart Institute, Bethesda, Maryland, Visiting Lecturer on Physiology, Harvard University, Boston, Massachusetts
- Henry D. Sayer, Former Research Associate, American Heart Association, Former New York State Industrial Commissioner, New York, New York
- Lamont R. Schweiger, M.D., Associate Clinical Professor of Medicine, Marquette University School of Medicine, Milwaukee, Wisconsin
- James J. Smith, M.D., Ph.D., Director, Department of Physiology, Marquette University School of Medicine, Milwaukee, Wisconsin
- Sidney S. Sobin, M.D., Ph.D., Research Professor of Internal Medicine, College of Medical Evangelists, Former Director, Work Classification Unit, Los Angeles, California
- David M. Spain, M.D., Associate Professor of Pathology, Columbia University College of Physicians and Surgeons, New York; Director of Laboratories, Beth El Hospital, Brooklyn, New York
- Donal R. Sparkman, M.D., Director, Cardiac Work Evaluation Clinic, and Associate Clinical Professor of Medicine, University of Washington School of Medicine, Seattle, Washington
- William H. Stewart, M.D., Assistant to the Surgeon General, U S Public Health Service, Washington, D C

- Henry Longstreet Taylor, Ph.D., Professor, Laboratory of Physiological Hygiene, University of Minnesota, Minneapolis, Minnesota
- Meyer Texon, M.D., Assistant Professor of Forensic Medicine, New York University Post-Graduate Medical School; Co-chairman, Subcommittee on Effect of Strain and Trauma on the Heart and Great Vessels, American Heart Association; Assistant Medical Examiner, New York, New York
- Harry E. Ungerleider, M.D., Director of Medical Research, Equitable Life Assurance Society, New York, New York
- L. E. Viko, M.D., Director, American Heart Association; Associate Clinical Professor of Medicine, University of Utah College of Medicine, Salt Lake City, Utah
- Maurice B. Visscher, M.D., Ph.D., Professor and Head of Department of Physiology, University of Minnesota School of Medicine, Minneapolis, Minnesota
- James V. Warren, M.D., Professor of Medicine, Duke University School of Medicine, Durham, North Carolina
- Theodore C. Waters, LL.B., Partner, Miles & Stockbridge, Baltimore Maryland
- Neill K. Weaver, M.D., Medical Director, Baton Rouge Refinery, Esso Standard Oil Company, Baton Rouge; Assistant Professor of Preventive Medicine and Public Health and Assistant Professor of Clinical Medicine, Tulane University School of Medicine, New Orleans, Louisiana
- Hans H. Weber, M.D., Director, Max Planck Institute of Physiology, Heidelberg, Germany
- Sidney B. Weinberg, M.D., Assistant Medical Examiner, City of New York; Assistant Professor of Forensic Medicine, New York University Post-Graduate Medical School, New York, New York
- Otto A. Wiesley, LL.B., Chairman, The State of Utah Industrial Commission and Utah Labor Relations Board, Salt Lake City, Utah
- Stewart Wolf, M.D., Professor and Head of Department of Medicine, University of Oklahoma School of Medicine, Oklahoma City, Oklahoma

PREFACE

The initial stimulus to the development of the First Wisconsin Conference on Work and the Heart came from a growing realization by several members of the faculty of the Marquette University School of Medicine that current knowledge concerning the influence of work, exercise, and stress upon the heart is inadequate, confused, and contradictory. Although this fact is probably most clearly demonstrated in the hearing room where in medical legal affairs one may hear widely divergent expert medical opinions, often unsupported by reliable scientific evidence, this is a much broader problem that affects all people at all ages, both those with heart disease and those with normal hearts. This matter concerns not only athletes and persons occupied in strenuous physical work, but housewives, children at play, and even individuals performing work usually classed as light or sedentary but associated with considerable emotional stress or tension.

In order to meet the need for more information in this field a conference was proposed at which experts in the area of work

and the heart would take up basic, fundamental considerations, bringing out the current level of our knowledge, pointing out the deficiencies in our information, and indicating the direction for future research and investigation. A sponsoring committee was formed which furnished invaluable guidance in the development of the meeting, the selection of the conferees, and the choice of subject matter. The Marquette University Medical School and the Wisconsin Heart Association agreed to support the conference jointly and the sponsorship of the American Heart Association, the National Health Institute, and the Industrial Health Council of the American Medical Association was readily forthcoming.

The Conference consisted of five panels dealing with separate aspects of the problems of the effect of work upon the cardiovascular system. These were basic physiology, clinical physiology, pathology, work classification, and workmen's compensation. The wide range from basic scientific problems to broad social applications was planned deliberately

so that all aspects of the field would be considered. Each panel was made up of ten to fourteen participants, each of whom had extensive experience in his field. Moderators and recorders directed the deliberations of each panel. To give breadth to the Conference, participants from Canada, England, Sweden, Finland, and Germany were invited. Each participant made one or more formal presentations, each of which was followed by extended discussion. The Conference was planned to allow time sufficient for almost unlimited discussion. The moderator of each panel prepared a summary that was presented at a final combined meeting of all the panels. Incorporated in these summaries were the accomplishments of the panel meetings and the major features of the discussions that occurred. The manuscripts of the formal presentations and the summaries of the moderators constitute the transactions published here. We have also published the discussion that followed the presentation of the final summaries, for this occurred on the one occasion when all the participants in the Conference met in a common scientific program.

The questions confronting the Conference were almost limitless in number. Many of these were presented to the moderators and conferees as the Conference was being planned. Some of them will be indicated here, as they were at the opening session, inasmuch as it was problems such as these that originally instigated the meeting. Does physical or emotional stress take its toll of the circulation and does it lead to an earlier onset of degenerative heart disease? Is the effect of work only evanescent and without permanent influence upon the heart? If heart disease is present, will effort or emotion aggravate the disorder, either acutely or over a period of time? If heart disease is present, is it possible that restriction of activity may have an adverse effect, both anatomically and psychologically? How does the heart do its work? Are there ways of modifying work loads or performing work so that the demands upon the heart are lessened? Do we have satisfactory techniques for measuring

the functional capacity of the normal or diseased heart and determining what the individual can do before he is assigned specific work? Do we have satisfactory techniques for measuring the demands made by specific jobs upon the heart? Work classification units have been established in many centers in the United States to aid in the rehabilitation and employment of patients with heart disease. At this Conference a large group of investigators working in these units was brought together for the first time. These workers were asked whether the salvage of patients in such units made worthwhile their expenditure of time and funds. Had they learned techniques or developed procedures that could be applied elsewhere in medicine and in the study and care of heart disease? Are workmen's compensation laws as currently administered in the United States adequate in the field of heart disease or are there deficiencies in their application? Have some states developed new procedures that have proved successful and worthy of broad application? Is it true that workmen's compensation insurance has become a form of health insurance in some states and may this militate against the person with heart disease who seeks employment?

The conferees who participated in this meeting came from the laboratory, clinic, workshop, and courts of law. The manuscripts presented here represent their answers to many of the questions such as those already outlined, with which they were confronted. To mention only a few, it will be seen that in the panel on basic physiology there were reports of significant observations of the chemical and physical phenomena associated with contraction and relaxation of the myocardium as it does its work. In the panel on clinical physiology there were reports concerning the influence of environment upon the work load of the circulation, the effects of very strenuous athletic competition upon the heart, and the demands of ordinary factory employment. In the panel on pathology there was an extensive interchange of ideas concerning the mechanisms causing coronary thrombosis and the demands made upon the

heart by stresses outside ordinary employment were evaluated. In the panel on work classification the gratifying results of careful evaluation of cardiac patients prior to their return to work were reported and the fact that patients with heart disease can work with safety was proved. In the panel on workmen's compensation there was a broad analysis of the current status of laws and procedures in this field, recommendations regarding medical testimony were made and new approaches to problems in this field that have been developed in some states were described.

As the Conference progressed it soon became clear that the answers to many of our questions are not forthcoming at this time. In some instances the techniques have not been developed with which to strike at the fundamental problems. The determination of the manner in which the heart does its work falls into this category. In some instances there has been a confusion of definitions and approaches. The measurements of physical fitness and of the functional capacity of the heart appear to fall into this category. In some fields there has been lack of basic information regarding the cause and course of the disease process. Acute coronary thrombosis and myocardial infarction are examples of this problem, as it will be seen that the panel on pathology was unable to give final answers to some of the most pertinent problems regarding the relation of stress to arteriosclerotic heart disease. In some areas work has been too recent to yield conclusive answers even though important knowledge may have been developed. Studies in the field of work classification reported at the Conference illustrate this.

Although this Conference closed with many of the original questions unanswered and an abundant yield of new questions,

those who developed the meeting feel it served a most worthwhile purpose. Active workers in several related fields were brought together for deliberation, an exchange of ideas, and mutual stimulation. There resulted an inventory and evaluation of the present status of the field and the many unsolved problems it presents. It is hoped that the Conference will act as a stone dropped into a quiet pool and that the resultant ripple will spread widely into many laboratories, clinics, hospitals, rehabilitation units, industries, and courts, bringing out the studies and observations urgently needed to solve our problems in the field of work and the heart.

The editors wish to acknowledge the grants from Wisconsin industries that made it possible to bring foreign participants to this meeting. Generous sums were received from the Western Printing and Lithographing Company, the Master Lock Corporation, the Allen Bradley Corporation, the Falk Corporation, Globe-Union Inc., and Lakeside Laboratories, Inc. The publication of these transactions was made possible by grant No. H-3145 from the National Institutes of Health. The editors are particularly indebted to Miss Mary Ferris and Mrs. Mary Beth Paape for their secretarial services and to Miss Mary Dougherty, Secretary of the Milwaukee Academy of Medicine, who assisted in the preparation of the bibliographies. Dr. Howard L. Correll of the Marquette University Medical School faculty was of great assistance in recording the discussions that developed at the final session of the Conference. Mrs. Eunice Stevens of Paul B. Hoeber, Inc. has been most helpful and cooperative throughout the compilation of the manuscripts and their preparation for publication.

FRANCIS F. ROSENBAUM, M.D.
Editor-in-Chief

INTRODUCTION

E. Cowles Andrus

The occasion for calling such a meeting as the First Wisconsin Conference on Work and the Heart is the recognition of a problem whose dimensions are too big to be ignored and the conviction that something, indeed, a great deal, can be done about it. Certainly no conference could be arranged under more favorable auspices. The sponsoring agencies, the Marquette University School of Medicine, the Wisconsin Heart Association, the American Heart Association, the National Heart Institute, and the Industrial Health Council of the American Medical Association include a wide variety of experience in this field and represent the expanding interest of thousands of lay and professional persons.

The concern of the public and of the medical profession has quite understandably been aroused by the startling annual mortality due to cardiovascular disease in this country—810,200 deaths in 1955, of whom 232,000 were under the so-called retirement age of 65. Only slightly more tardily have we awakened to the fact that a far larger segment of the population, approximately 10,000,000 persons (1 out of every 16), is currently liv-

ing with heart disease. Diseases of the heart and circulation cause the loss of about 150 million man days of work each year and an annual economic loss of two and a half billion dollars. The federal tax on this amount at ordinary rates would be nearly 300 million dollars. Men and women at the peak of their skill and productivity are lost to industry, to their families, and to the nation. Moreover, if present trends persist and means of therapy continue to improve the number of those who survive though afflicted will certainly not diminish.

I have been asked to express a keynote for this conference. In considering what I might say, my mind turned back to some remarks made by Sir James Mackenzie in 1921 referring to his early experience in medical practice in the 1880's and concerning the heart and another sort of labor.¹

There are few subjects in medicine of which an accurate knowledge is more urgently required than that of a woman's fitness for child-bearing. That a great strain is thrown upon the maternal heart during pregnancy and labour is well known. It is a matter of experience, too, that the preg-

nant state may throw more work on the heart than it can bear, with the result that the mother's life is lost, or her health irreparably impaired. The child, too, often dies. These disastrous happenings surround a natural process with dread and mystery, for the dread is aggravated by the fact that the source of danger is not clearly realized. As a result all sorts of signs are looked upon with suspicion—signs innocent as well as signs grave. Needless to say, this obscurity does a great deal of harm. Many women are subjected to unnecessary alarms and restrictions when pregnant, others have to suppress the natural desire of motherhood; while connubial relations are often disturbed by warnings and hints of dangers which may never arise.

This unsatisfactory state of affairs is entirely dependent on a defect in medical knowledge which it is quite possible to remedy. That the matter is not worse is in a measure due to the fact that the vast majority of women, especially women of the working-classes, never have their hearts examined. Were they systematically examined, abnormal signs would be discovered in many instances, the significance of these signs would not be understood, grave doubts would be aroused. Thus, many who have passed unscathed through the pregnant state would have been unnecessarily frightened and alarmed. Before the discovery of auscultation and the use of the stethoscope, about 100 years ago, experienced physicians estimated the state of the heart by its manifest functional efficiency. There is no doubt in my mind that the line pursued by those physicians was sounder than that which has been followed since the introduction of mechanical devices. The coming of the stethoscope was the beginning of a method in clinical research which has greatly hampered the practice of medicine, in that the introduction of mechanical methods has led to confusion as to the kind of knowledge which these methods are capable of affording.

One unfortunate consequence of this concentration upon the new method, however, was to give it a preponderating importance in the examination of the heart, and to minimize the value of the study of other phenomena, with the result that unjustified conclusions were drawn from mere modifications of the sounds. This, as has been indicated, was an error inherent in the view taken, at that time, of the possibilities of the new instrument. The physician who detected an abnormal sign supposed that in detecting it he had obtained information of sort hitherto only

obtainable by a test of functional capacity. He believed that he had gained knowledge entitling him to make a prognosis without the labour of determining how far the patient's activities were already curtailed.

Today no thoughtful person would deny the usefulness of the stethoscope or of the even more refined instruments which the physician has at his disposal to discover and to measure abnormalities of the heart and circulation, but Mackenzie's insistence upon the importance of functional capacity is as pertinent today as it was when he wrote these words. Moreover, I venture the opinion that no device has yet been designed which is uniformly more valuable in estimating the patient's capacity to do a job than his own description of his sensations or symptoms obtained by an unhurried and careful interview.

In a very real sense, concern for the working patient with cardiac disease has been at the center of the Cardiac Clinic movement in this country. As will be pointed out elsewhere in this conference, this began with the organization by Dr. H. V. Guile of an evening Cardiac Clinic at Bellevue Hospital in New York City in 1911. This enterprise and interest extended to what has later become the New York Heart Association and the American Heart Association. Since the reorganization of the American Heart Association after World War II, activities on behalf of the cardiac in industry have grown rapidly and fruitfully. Much has been done and much more endeavored to provide a useful estimate of the fitness of those afflicted with cardiac disease in order to permit their return to work without accelerating circulatory failure. Behind this there is a continuing effort directed at the control of the primary disease, the prevention of recurrent disease or breakdown, and the treatment and rehabilitation of those in whom cardiovascular disease has produced disability.

This conference is in reality a collective exercise in prognosis, bringing together those experienced in basic or clinical physiology or pathology, and in the current techniques of

measuring functional capacity and, no less important, those familiar with the problems of workmen's compensation and with the impediments in our present laws and policies of insurance as they affect a disabled patient attempting to return to gainful occupation. To predict the content of our discussions here during the next three days or to forecast the conclusions that will emerge would be to compose a summary before the book is written. However, one can assume with assurance that we shall learn from each other, both regarding what is known and what is not known, and that we shall return to our lab-

oratories or clinics or offices impressed anew with the challenge of this problem of putting the person with heart disease to work. Your presence here attests your interest and your optimism.

"Rejoice also in tribulation; knowing that tribulation worketh patience; and patience, experience; and experience, hope:" Romans 5:3, 4

REFERENCE

1. MACKENZIE, JAMES: *Heart Disease and Pregnancy*, London, Oxford Medical Publications, 1921

PART I BASIC PHYSIOLOGY

CHAPTER 1 THERMODYNAMICS OF MUSCLE

Robert W. Ramsey

In respect to thermodynamic analyses of muscular contractions, one might arbitrarily divide physiologists into two broad groups. One group endeavors to determine the fundamental properties and thermodynamic cycles of the muscle machine by studying models which in varying degrees have some resemblance to the living muscle machine. This group includes so many distinguished investigators that it would be invidious to mention any without including all, this task I will leave to Dr. H. H. Weber, who is of this group and is participating in this Conference. Basically, the hope of this latter group is to begin with the elemental building blocks and reactions and gradually determine the intermediate steps that lead to a complete understanding of the final complex living muscle machine.

The second group of physiologists study the behavior of living muscle. All this group are well aware that their investigations will not provide the ultimate explanation of the mechanisms of muscle contraction but feel that the gap between the model experiments and the living machine is so great that in the

interim a rough thermodynamic analysis is justified, if for no other reason than as a guide to future experiments.

This latter approach has a very ancient history in contrast to the relatively recent model approach. As early as the seventeenth century, Borelli proposed that a muscle at rest might be likened to an extended spring, which upon contraction expended its energy in doing work and that during relaxation this energy was restored. This clearly is a thermodynamic cycle, though this terminology did not come into use until the nineteenth century. You will recall that A. V. Hill received the Nobel Prize for demonstrating this cycle, based upon his measurements of the isometric length-tension diagram and the heat liberated during muscular contractions. The key features of this cycle at the time were that, in contraction, the muscle liberated all the energy stored during relaxation, so that this energy had to appear as external work or as heat in overcoming viscosity.

The first major assault on the Hill cycle stemmed from Fenn's¹ experiments done in Hill's laboratory, on the energy balance in

isotonic contractions Fenn showed clearly that when an isolated muscle is allowed to do external work, an extra amount of energy is liberated during contraction exactly equal to the work done. From these experiments, Fenn argued that energy for contraction is supplied from chemical stores during the contraction phase itself. This cycle may be called an active contraction—passive relaxation cycle.

While maintaining that his cycle was correct, Hill² was led to the conclusion that most of the energy expended by a runner running at constant velocity was wasted as heat in overcoming the viscosity of his muscles. This was rather soon shown to be fallacious by Fenn,³ who demonstrated that if one added up all the work done in running in accelerating and decelerating the limbs, raising and lowering the center of gravity, overcoming wind resistance and ground reactance, that the total work done left very little energy to be used in overcoming viscosity. Later Fenn and Marsh⁴ showed on isolated muscle that the force-velocity relation of muscle was not linear (as it should be on a Newtonian basis of viscosity) but approximately exponential.

With this demonstration, Hill⁵ began a thorough investigation of force-velocity and energy balance studies of isotonic contraction with greatly improved myothermic techniques. In a series of beautiful papers Hill confirmed Fenn's³ earlier observations and succeeded in linking the force-velocity data with the heat data in the form of an empirical equation now called Hill's *characteristic equation*. Hill thus adopted completely Fenn's point of view, namely, that energy is supplied as needed from chemical stores during contraction and that relaxation is purely passive.

During this period, Mrs. Ramsey and I⁶ were studying the behavior of single muscle fibers in Fenn's laboratory. Gradually we discovered a number of phenomena, some of which had been noted by others before working on whole muscle, but whose results were not generally accepted, which threw some doubt on the Fenn-Hill hypothesis. The orig-

inal papers would have to be consulted for the details but some of the phenomena we noted that are inconsistent with the Fenn-Hill hypothesis are:

1. A single muscle fiber actively and vigorously relaxes. The mechanism of this active relaxation may be open to question but the phenomenon is not. We believe we have adequate evidence to show that this active relaxation is not due to sarcolemmal forces.

2. We were never able to fatigue a single muscle fiber in the sense of a diminished contractile response, "fatigue" was simply a failure of the excitatory mechanism whether stimulated directly or indirectly through its nerve.

3. The length-tension developed diagram (tetanic) is virtually invariant with temperature.⁷

Taken at face value, these three phenomena are inconsistent with the Fenn-Hill hypothesis but consistent with Hill's earlier hypothesis, provided the heat he formerly identified as frictional loss is now recognized as his *shortening heat*. Encouraged by Drs. Harry Blair and Fenn, I re-examined the cycle that presupposed that energy was previously stored in the contractile machinery, released during contraction, and restored from chemical stores during relaxation. The two chief hurdles that had to be surmounted were the old one of the Fenn effect and a new one that had not been recognized before, namely, that if this cycle were correct, the force-velocity relation necessarily had to be related to the length-tension diagram. The original analysis⁸ would have to be consulted for details, but it will be sufficient here to note that it adequately, if empirically, accounted for the energetics of muscle contraction for all lengths equal to or less than rest length. Further, purely on the basis of energetics, this cycle predicted that if a muscle shortened more than a third of its rest length, it would have insufficient energy actively to relax. Since we had previously found that when a single muscle fiber shortens more than a third of its length, active relaxation is abolished, I felt that this constituted rather strong evidence in support of this cycle. In general, in applying antithetic

cycles to phenomena where all the details cannot be specified, the best that can be demonstrated is that a particular cycle is adequate. If, however, of two antithetic cycles, failure occurs in one part of the cycle, it will in general lead to opposite phenomena so that the failure of active relaxation occurring where postulated, lends strong support to the cycle.

Buchthal⁸ and later Polissar¹⁰ approached the thermodynamic analysis of muscle contraction from the viewpoint of reaction rate principles in which they assumed that the ultimate contractile unit existed in a long (L) or short (S) state and that the state of the contracted muscle could be represented by the statistical distribution of numbers of units going from long to short or short to long at any given instant. Both of these papers were very stimulating but their chief defect, as I see it, is an inadequate accounting for the Fenn effect. Both have the distinct merit, however, of offering a basis for speculation about the behavior of muscle at lengths greater than rest length.

Stimulated by these latter papers and by an observation of Wilkie,¹¹ I re-investigated the cycle I had previously shown to be adequate, using reaction rate principles and drawing an analogy between the behavior of muscle and the polymerization of some high polymers. This analysis¹² provided a more elegant demonstration of the adequacy of the cycle and introduced the concept that the series elastic element might well be due to the isolation of reactive groups during shortening.

While I have chosen to present this in a very personal way, I do not wish you to carry away the impression that I firmly believe in the active relaxation cycle as the final answer, for I do not. I do believe that with the evidence at hand it is the more likely one and certainly that it is adequate. Also, the implicit assumption that the energy released by a muscle is derived from internal energy changes rather than entropy changes may be partly, but certainly not wholly, wrong.

If the above were all that was known

about skeletal muscle, we could afford to be somewhat complacent about our thermodynamic knowledge, for in other fields an equivalent body of data has enabled great extension of knowledge by thermodynamic reasoning. Our complacency is completely shattered if we consider the phenomena of muscle at lengths above rest length and muscle fibers in the delta state. For the latter we know three facts: that active relaxation is abolished, tetanic tension is half the normal fiber at equivalent lengths, and that the rate of development of tension is slow.

Many intriguing questions remain to be answered about the delta state. To name two, is the Fenn effect abolished in the delta state? My guess is that it is. Can a muscle in the delta state accumulate an oxygen debt or does it behave like heart muscle, which can accumulate only a very small oxygen debt?

When a resting muscle is stretched beyond its resting length its resting metabolism is greatly increased^{13,14} and its surface pH shifts in the direction of alkalinity.¹⁵ When stimulated at such an extended length, it develops tension slowly and to a lesser degree than at rest length. From single muscle fiber studies we know that the refractory period increases with stretch but that the basic time constant of the membrane remains unchanged. This latter probably means that the capacity per unit area of the membrane increases, but that the resistance diminishes in proportion. In turn this would necessitate a speeding up of the metabolic pump to keep the membrane charged to its normal state, and the shift of pH toward alkalinity would be the mechanism by which the increased oxygen consumption was brought about, since the oxidative enzymes would be poised more on the oxidative side.

It has long been known that the cost of doing *negative* work, that is, bodily movements that involve stretching a muscle while it is contracting, is much less than the cost of doing *positive* work, but only recently has it been realized how great the difference may be. For example, Abbott¹⁶ found that the cost of doing negative work could be as much as six times smaller than doing positive work.

Does stretching a contracted muscle reverse the machine or is it merely a decrease in the number of fibers contracting? Some of Fenn's¹ and Hill's⁵ heat measurements suggest the former may be the case.

These facts are put forward merely to place in perspective how little is our knowledge of the most thoroughly studied tissue in physiology, isolated skeletal muscle. When we consider the function of skeletal muscle in voluntary movements in the body, for practical purposes three quarters of the properties that appear important in isolated tissue are rendered unimportant, because of the ability of the central nervous system to dole out excitation to more motor units or at a greater or lesser frequency, or to kick in an antagonist at the proper moment. It would be foolish to claim that the properties of isolated muscle play no role in voluntary behavior, for they do, but as an example of what I mean, the force-velocity curve of a human forearm to me is chiefly an expression of the innervation pattern of that individual, not the force-velocity relation of the isolated biceps, though this will be the limiting relation, particularly when motivation is extreme.

In this respect the heart is very different. While its rate of beating and the force it exerts in contraction may be modified by many factors, if we consider any one beat at the instant the heart starts contracting the resultant pressure and volume changes are solely dependent on the contractile power of the whole tissue itself. Thus, there is every reason to expect that the establishment of a basic thermodynamic cycle for isolated heart tissue will have more immediate application to the intact organism than does the establishment of such a cycle for skeletal muscle have to our ordinary voluntary movements. The fuel that is burned may be very different in the intact versus the isolated preparation for either heart or skeletal muscle, as Drs. Visscher, Bing, Andres,^{17,18,19} and others have shown, but the cycle would not be, though part of the restorative energy might be supplied from other organs. The difficulties of establishing such a cycle for heart tissue are tremendous, as everyone knows.

After all, if it had not been for the kindness of nature, who gave us the frog sartorius muscle, I would wager our knowledge of the energy balance of skeletal muscle would be where our knowledge of the energy balance of heart muscle is today.

BIBLIOGRAPHY

- 1a. FENN, W. O.: Quantitative comparison between energy liberated and work performed by isolated muscle of the frog, *J. Physiol.* 58:175, 1923.
- 1b. FENN, W. O.: Relation between work performed and energy liberated in muscular contraction, *J. Physiol.* 58:373, 1924.
2. HILL, A. V.: *Muscular Movement in Man; the Factors Governing Speed and Recovery from Fatigue*. New York, McGraw-Hill Book Co., 1927.
- 3a. FENN, W. O., and MORRISON, C. A.: Frictional and kinetic factors in work of sprint running, *Am. J. Physiol.* 92:583, 1930.
- 3b. FENN, W. O.: Work against gravity and work due to velocity changes in running, movements of center of gravity within body and foot pressure on ground, *Am. J. Physiol.* 93:433, 1930.
4. FENN, W. O., and MARSH, B. S.: Muscular force at different speeds of shortening, *J. Physiol.* 85:277, 1935.
5. HILL, A. V.: Heat of shortening and dynamic constants of muscle, *Proc. Roy. Soc., London, B*, 126:126, 1938.
- 6a. RAMSEY, R. W., and STREET, S. F.: Isometric length-tension diagram of skeletal muscle fibers of frog, *J. Cell. & Comp. Physiol.* 15:11, 1940.
- 6b. RAMSEY, R. W., and STREET, S. F.: The relation of the absolutely refractory period, relatively refractory period and tension in isolated muscle fibers of the frog, *Am. J. Physiol.* 133:419, 1941 (*Proc. Am. Physiol. Soc.*).
- 6c. RAMSEY, R. W., and STREET, S. F.: Absence of fatigue of the contractile mechanism in single muscle fibers, *Fed. Proc.* 1:70, 1942.
- 6d. RAMSEY, R. W., and STREET, S. F.: Muscle function as studied in single muscle fibers, *Biol. Symposia* 3:9, 1941.
- 6e. RAMSEY, R. W.: Muscular contraction, dynamics of single muscle fibers, *Ann. New York Acad. Sci.* 47:675, 1947.
7. WASHINGTON, M. A., and ARRIGHI, M. S.,

- STREFF, S F, and RAMSEY, R W.: Q10 of the maximum tetanic tension developed by isolated muscle fibers of the frog, *Science* 121 415, 1955
- 8 RAMSEY, R. W. "Muscle-physics," in: O GLASSER (Ed.), *Medical Physics* Chicago, Year Book Publishers, 1944, p. 784
- 9 BUCHTHAL, F., KAUER, E., and ROSEN-FALK, P. The rheology of the cross striated muscle fibre, with particular reference to isotonic conditions, *Kgl Danske Videnskab Selskab Biol Medd* 21, no 7, 1951
- 10 POISSAR, M J. Physical chemistry of contractile process in muscle, *Am J Physiol* 168 766, 1952
- 11 WILKIE, D R. Facts and theories about muscle, *Progr Biophys and Biophys Chem* 4 288, 1954
- 12 RAMSEY, R W. Analysis of contraction of skeletal muscle, *Am J Physiol* 181 688, 1955
- 13 MEYEROF, O., GEMMILL, C L., and BENETATO, G. Ueber den isometrischen Koeffizienten des Sauerstoffs normaler und jodtrugare-vergifteter Muskeln, *Biochem Ztschr* 258 371, 1933
- 14 FENG, T P. Thermo-elastic properties of muscle, *J Physiol* 74 455, 1932
- 15 DUBUISSON, M. Recherches sur les modifications de pH et sur les changements du point isoionique de la myosine pendant l'excitement et la contraction du muscle, *Arch internat. physiol* 50 203, 1940
- 16 ABBOTT, B. C., BIGLAND, B., and RITCHEY, J M. Quoted by HILL, A. V. Mechanics of voluntary muscle (Lady Jones Lecture), *Lancet* 2 947, 1951.
- 17 BARNES, R H., MACKEY, E M., MOF, G K., and VISCCHER, M. G. Utilization of beta hydroxybutyric acid by isolated mammalian heart and lungs, *Am J Physiol* 123 272, 1938
- 18 BING, R J., SIEGEL, A., VITALE, A., BALBONI, F., SPARKS, E., TAESCHER, M., KLAPFER, M., and EDWARDS, S. Metabolic studies on human heart in vivo, studies on carbohydrate metabolism of the human heart, *Am J Med* 15 284, 1953
- 19 ANDRES, R., CADER, G., and ZIFRLER, K L. The quantitatively minor role of carbohydrate in oxidative metabolism by skeletal muscle in intact man in the basal state, measurements of oxygen and glucose uptake and carbon dioxide and lactate production in the forearm, *J Clin Invest* 35 671, 1956

CHAPTER 2 MOVEMENTS AND CONTRACTILE PROTEINS OF MUSCLES AND CELLS

Hans H. Weber

ATP REQUIREMENT AND RESYNTHESIS

Nowadays we know that the energy set free by foodstuffs serves the sole purpose of forming and restoring ATP (adenosine triphosphate). This was one of the extraordinary discoveries that biochemists made during the first half of our century. The energy stored in the ATP is needed for the work done by our tissues. In order to liberate this energy the ATP must be split.

Our problem is to investigate how the energy yielded by the ATP splitting is used to produce the movements of muscles and cells. It is difficult to obtain the answer for the ATP content of cells and muscles is very low. It is sufficient for only 20 to 30 single twitches of the skeletal muscles and is, therefore, scarcely sufficient for a tetanus of one second's duration. Nevertheless, our muscles are able to exert tetanic contractions of a much longer duration. This is because during muscular work the ATP is restored almost as rapidly as it is broken down. The chemical and energetic means employed in this res-

toration process originate from the intermediary metabolism of our foodstuffs and in addition from certain special substances, such as the different phosphagens, which contain energy-rich phosphate bonds.

The simultaneousness of ATP requirement and ATP resynthesis means two important things. (1) A decrease in the ATP level seldom reveals an increased ATP breakdown, e.g., such as occurs in connection with muscular work, (2) The various reactions that lead to resynthesizing ATP occur concurrently with, and immediately after, the ATP breakdown.

If we now consider, moreover, that a few thousandths of a second before the contraction process proper, the electrical and chemical processes of excitation occur on the muscular membranes, it seems almost impossible that we should be able to detect from this wealth of almost simultaneous events the very process by which the chemical energy is converted into mechanical energy. We are therefore not surprised that for 20 years it

has been undecided whether the energy set free by the splitting of ATP is transferred directly or indirectly to the mechanically active protein

ISOLATION OF CONTRACTILE PROTEINS

In the main, therefore, a new approach has been made recently toward investigating animal motility. Research has attempted to isolate from all kinds of living cells and tissues the protein structures that are responsible for motility, with the purpose of retaining them in such a state that they could function as if they were alive. This has been achieved in three different ways, either mechanical, functional, or chemical.

1. By mechanical isolation, muscle fibrils of a thickness of 1μ can be obtained from all cross-striated muscles.¹ These myofibrils represent the contractile apparatus of the striated muscle fibers free from other proteins

2. A functional isolation signifies that the active protein structures remain in the non-muscular cell² as well as in the muscle fiber.³ The cell membranes, however, are destroyed in a suitable manner, and at the same time all metabolic substances, soluble enzymes, ions, and so on, are extracted

3. A chemical isolation is performed by extracting and purifying the contractile proteins from muscles and nonmuscular cells. The contractile proteins are subsequently precipitated in physiological salt solutions into fine protein threads. The protein molecules are then aligned in parallel, corresponding to the alignment that they originally had in the muscle fibers. Since all these preparations are dead and do not contain any autolytic enzymes, they can be stored in ice-boxes for months

If the right metabolic substance is added to the preparations, they exhibit the very movements (contraction and relaxation) that these structural proteins exhibited during life. In spite of the fact that these preparations are contractile structures functioning like living structures, they are designated *models* and characterized according to the procedure of preparation: (1) fibril models when myo-

fibrils are isolated mechanically, (2) fiber models or cell models when a functional isolation by cytolysis has taken place, and (3) thread models when threads are prepared from chemically isolated proteins

BEHAVIOR OF CONTRACTILE PROTEINS

All types of muscle models, i.e., fibril models, fiber models, and thread models shorten, when they are in neutral solutions of physiologic ionic strength in the presence of magnesium, as soon as ATP or another nucleoside triphosphate is added. The same applies to models that are prepared from expanded amoeboid cells. On adding ATP they each pull in their pseudopodia and contract into a ball-like shape (Fig. 2-1). Models prepared from cells that were in the telophase stage of mitosis contract the cell equator until the halves are connected by just a thin thread (Fig. 2-2). For all these models the optimal ATP concentration is identical and corresponds rather well to the physiologic concentration of ATP. In all models both ATP-splitting and ATP-induced contraction are inhibited by the same poisons, for instance, by mersalyl or Ba Suramin.^{4,5,6,7,8} Neither of these latter poisons, however, completely inactivates the majority of the other ATP-splitting enzymes contained in tissues.⁹ The magnesium requirement, the dependence on temperature, and certain other qualities



FIG. 2-1 Contraction of cell models (amniotic fibroblasts) by 2×10^{-4} M ATP at pH 7.0, ionic strength 0.14μ , $T = 37^\circ\text{C}$. 28-day-extraction. (Left) Without ATP (Right) 12 min after the addition of ATP (Hoffmann-Berling⁴)

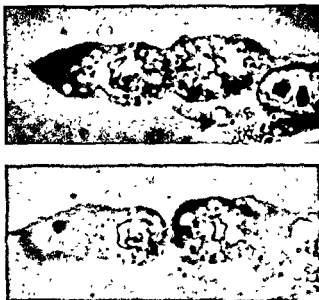


FIG 2-2. Cytokinesis of the telophase model (subcutis fibroblast) by $2.5 \times 10^{-3}M$ ATP at pH 7.4, ionic strength 0.12μ ; $T = 22^\circ C$; 2-h-extraction. (Above) Without ATP (Below) 12 min after the addition of ATP (Hoffmann-Berling³).

characteristic of contraction, are also identical for both muscle models and the cell models described^{4,10}

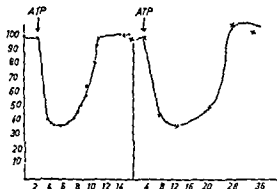
The chemically purified contractile proteins of muscles and single cells are identical in all qualities studied so far.⁹ Floccules precipitated from the purified contractile protein of sarcoma cells contract in the same way as actomyosin floccules (Fig 2-3) or as the entire cell model prepared from sarcoma cells. The solubility of actomyosin, the contractile protein of mammalia, depends both

on ionic strength and on ATP concentration in the same way as does the solubility of the contractile cell protein of sarcoma cells. Both proteins immediately split the added ATP and show an identical dependence on the pH, if magnesium is present. Both proteins no longer split if mersalyl in the same low concentration ($\sim 10^{-4}M$) is added^{4,5,6,7,8,9}. On addition of ATP solutions both kinds of proteins show in solutions the same reversible decrease in viscosity (Fig. 2-4). Therefore it is reasonable to assume that the cause of the



FIG 2-3. Shrinking of gel particles precipitated from the solution of purified contractile protein prepared from sarcoma cells (Left) Without ATP. (Right) 45 min after the addition of $5 \times 10^{-3}M$ ATP, the half on the right figure shows the increase in the density of the particles $T = 22^\circ C$; pH = 6.9; ionic strength = 0.08μ , (Hoffmann-Berling⁹).

Fig 2-4 Comparison between the changes in viscosity of an actomyosin solution from rabbit musculature and of a solution of the contractile protein from sarcoma cells on the addition of ATP. Ordinate: specific viscosity in percentage of the initial viscosity Abscissa: time in minutes (Left) Actomyosin solution, redrawn according to Kuschinsky and Turba" (Right) Solution of sarcoma protein, redrawn according to Hoffmann-Berling.* 10⁻⁴M MgCl₂, pH ~ 7, ionic strength ~ 0.6 M, T = 20°C, ATP on the left 2 × 10⁻⁴M, on the right 1 × 10⁻⁴M



lowering in viscosity is likewise identical for both proteins. This means that the cell protein like actomyosin is a dissociable complex of two proteins.

The only difference is that the ATP is split by the contractile cell protein about 200 times more slowly than by the protein of skeletal muscles (Table 2-1) but the contraction of both the cell model and the living protozoan is similarly a few hundred times

slower than the contraction of the living skeletal muscle and its models.

If ATP is present but not split by the contractile structures,** cell models as well as all kinds of muscle models relax. In these experiments it does not matter whether the splitting is inhibited by a reversible poisoning, for instance, by mersalyl (Hg-salicyl-allylamide-sodium acetate) (Fig 2-5) or by a physiological factor,¹³ which is found in the granules of the muscle (Fig 2-6). If the ATPase activity of the contractile structures is inhibited, the models relax, in spite of the fact that the other cellular and muscular

TABLE 2-1 RATE OF ATP SPLITTING AND AMOUNT OF CONTRACTILE PROTEIN (According to H. Hoffmann-Berling*)

Prepared from	μ moles P Mg protein \times min	Mg. contractile protein 100 mg tissue
Skeletal muscle	0.35	~10
Yoshida-sarcoma	0.003	~0.2

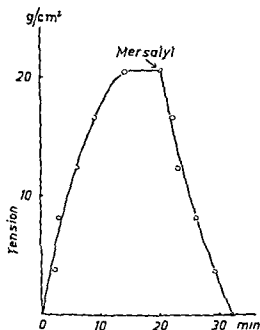


FIG 2-5 Relaxation of a contracted film, prepared from extracted fibroblasts, produced by the addition of mersalyl, tension produced by ATP, dropped to zero by the addition of 2 × 10⁻⁴M mersalyl, T = 22°C, pH = 6.8, ionic strength = 0.08 M (Hoffmann-Berling*)

ATPases remain active (Fig 2-7). This observation makes it evident that not ATP splitting in general but ATP splitting induced only by the contractile structure produces contraction. On the other hand, upon the complete removal of ATP cells and muscle models become rigid.* This is similar to the behaviour of the intact muscle, which passes into rigor mortis when its ATP content is destroyed by autolysis.

Hence, ATP reacts with the contractile

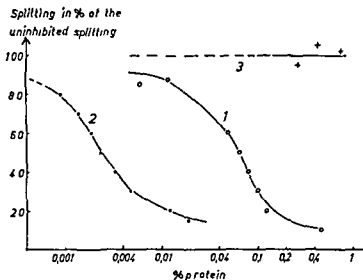


FIG 2-6 Binding of relaxing factor to muscle granules Dependence of ATPase activity of fibrils on the protein concentration (1) of the extract containing granules prepared from muscle, (2) of the granules isolated from the extract, (3) of the extract after the removal of the granules Ordinata ATP splitting by fibrils as percentage of normal splitting Abscissa concentration of the tested protein in percentage, ionic strength $\sim 0.14 \mu$, pH ~ 7 , $\sim 4 \times 10^{-4} M$ ATP (Portzehl¹⁹)

protein in two different ways: (1) If it is split by these proteins, it produces contraction, (2) if it is not split by them, but bound to them, it induces relaxation and renders the otherwise rigid structures plastic This applies to the above mentioned cell movements and to every kind of muscle In these

interactions ATP reacts with the contractile proteins directly Even for the ATP splitting no special enzyme is interposed

Hence, by means of the so-called model technique it is well established that on the one hand, with ATP and a few related nucleoside triphosphates and, on the other,

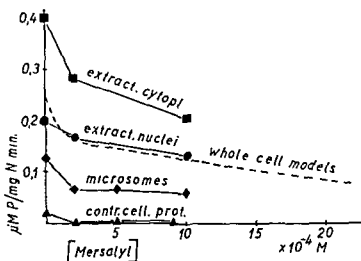


FIG. 2-7 Mersalyl sensitivity of cell models and isolated fractions prepared from cell models Ordinata ATP splitting in moles per mg protein \times minute $T = 22^\circ C$, pH $= 6.8$; ionic strength $= 0.08 \mu$, $10^{-4} M$ ATP. (Hoffmann-Berling⁹)

with the inhibiting factor for ATP splitting found in the sarcosomes, the contractile proteins in every stage of phylogenetic development are able to contract or relax

A SPECIFIC REACTION

It is very unlikely that the contractile proteins would *not* make use of the capacity to react in the way described during life. It becomes still more unlikely when we consider the extraordinary specificity of these reactions: *no* isolated *contractile* structure contracts, if any other substance of the intermediary metabolism is added instead of ATP or related nucleoside triphosphates. And, vice versa, ATP induces no other protein structure, neither a natural nor an artificial one, to contract except the contractile structures of cells and muscles.

All details of the contraction *in vivo* are qualitatively quite identical and quantitatively almost identical with those of the model con-

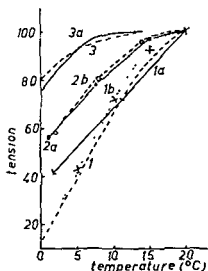


FIG 2-8. Dependence of tension on temperature.¹⁰ Curve 1 tetanized diaphragm (rat, Hajdu). Curve 1a fiber model (rabbit, A Weber). Curve 1b actomyosin thread (rabbit, Portzehl); both in $3 \times 10^{-4}M$ ATP. Curve 2a fiber model (smooth muscle, Anodonta, G Ulbrecht and M Ulbrecht) in $2.5 \times 10^{-4}M$ ATP. Curve 2b actomyosin thread (smooth muscle, Anodonta; Durr and Portzehl) in $1.5 \times 10^{-4}M$ ATP. Curve 3 tetanized frog muscle (A V Hill). Curve 3a muscle in thawing contraction (frog, Hajdu).

TABLE 2-2 ACTIVE MAXIMAL TENSION (kg/cm^2)¹⁰

Kind of muscle	Fiber model	Living
Skeletal muscle		
Rabbit	4	5
Frog	3	2
Smooth muscle		
Anodonta adductor		
yellow	5 1	4.5
white	2 1	
Cow rectum (longitudinal muscle)	0.6	0.7

traction.⁶ An impression of the qualitative agreement can be obtained from the film, "Movements of extracted cells and muscle fibrils (*models*) induced by adenosine triphosphate (ATP)."¹¹ The quantitative agreement has been ascertained by comparing muscles and muscle models. All muscle models develop isometrically the same maximum tension as the living muscle from which the model concerned has been prepared (Table 2-2). This is the case, even though the maximum tension of the living skeletal muscle of the rabbit is ten times as great as the

isometric tension developed by the longitudinal musculature of the cow rectum. The maximum tension developed by models and muscles is equal not only at room temperature but at all temperatures down to 0°C, although the dependence on temperature is extremely different for cold-blooded and warm-blooded animals (Fig 2-8). Under isotonic conditions muscles and models shorten by 75 to 85 per cent of their standard length.¹⁰ However, the living *skeletal* muscle shortens to such an extent only when it passes into a special state, i.e., into Ramsey's Δ state.

Not only are the degree of maximum tension and the extent of maximum shortening developed by muscles and models and their dependence on temperature identical, but so are most of the characteristic details of contraction, such as Hill's quick-release phenomenon, which exhibits the following peculiarity. If a muscle or a model in isometric

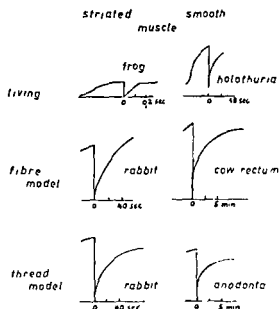


FIG 2-9 Tension after quick release.¹⁴ Release 10 per cent of relative length Frog muscle (Gasser and Hill); body wall muscle of holothuria (Hill), fiber model prepared from rabbit (A Weber), fiber model prepared from cow rectum (G Ulbrecht and M Ulbrecht), thread prepared from a solution of rabbit actomyosin (Portzehl), thread prepared from a solution of Anodonta actomyosin (Dorr and Portzehl)

contraction is allowed to shorten freely by 5 to 10 per cent, the tension falls to zero at the moment of shortening and rises again, shortly afterwards, to the equilibrium tension of the new length (Fig 2-9). This phenomenon has been observed nowhere else except during the contraction of the living muscle and during that of muscle models. During the shortening of both the living muscles and the models birefringence decreases, the amount of decrease being the same for fiber models and living muscles.¹² As a final example I want to mention that the proportion of the total energy transformed during the contraction phase into mechanical work by either the living muscles or their models is identical or practically so.¹⁵

We are prompted to conclude from all these findings that the ATP mechanism is identical in muscles and models and that it was provided by Nature even in the developmental stage of the Protozoa.

OTHER MECHANISMS OF MOVEMENT

Protozoa are endowed with further mechanisms of movement. During the anaphase of mitosis the spindle body or "Stemmkoerper" elongates and pushes the two sets of chromosomes apart to the poles of the division spindle. The same occurs as soon as ATP in a physiologic salt solution and in a physiologic concentration is added to a cell model that has been prepared from a cell in the early anaphase (Fig. 2-10). However, this effect of ATP is due to a different mechanism, for the action of ATP in producing this effect is *not* inhibited by mersalyl poisoning.¹ The same effect can also be produced by pyrophosphate, though less effectively.¹ It may be mentioned that the ATP cannot be replaced by pyrophosphate to produce muscle contractions or the musclelike kind of cell contractions.

Still, there is another type of contraction and another type of elongation exhibited by organelles of certain cells. Neither can be produced by ATP but can be produced instead by calcium (Ca) ions. Models prepared from stalks of *Vorticella* contract actively, developing substantial tension, if Ca

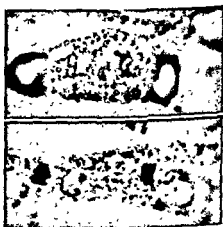


FIG 2-10 Chromosome sets of a cell model obtained from extracted subcutis fibroblasts moving apart during the anaphase of mitosis. Ionic strength = 0.22 M, pH = 7.4; T = 22°C (Above) Before adding ATP. (Below) 10 minutes after the addition of ATP (Hoffmann-Berling¹).

ions are added (Fig. 2-11), whereas addition of ATP causes relaxation. Isolated trichocysts of *Paramecia* elongate to very fine needles in a flash if, in the absence of ATP, Ca is added, whereas in the presence of ATP this Ca-induced elongation is inhibited.¹⁸ Nature, it seems, is still making experiments with the organelles of the Protozoa concerned with movement. For the muscles, however, the organs of movement of the Metazoa, Nature has continued to use only her best discovery, the ATP-induced contraction cycle.

SLIDING OF FILAMENTS VERSUS ELASTICITY

It is important to know how the ATP splitting induces the ATP contraction of cells



FIG. 2-11 Ca-induced contraction of an ATP-free model from the stalk of *Vorticella gracilis*. The dark line inside the stalk is the contractile thread, the lighter border is the elastic sheath. (Left) Before the addition of Ca. (Middle) On Ca addition the contractile thread has contracted the sheath into a screwlike shape. (Right) On contraction of the contractile thread has torn itself apart, the sheath has lengthened and has pushed the dark fragments apart. A comparison between the picture to the left and that on the right shows the amount of shortening of the contractile thread (Hoffmann-Berling¹⁸).

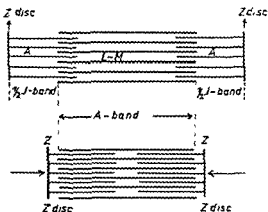


FIG. 2-12 Diagram of two sarcomeres (J. Hanson and H. E. Huxley¹⁹), simplified. A = actin, LM = L-myosin. (Above) Stretched. (Below) Contracted.

and muscles. We know now from the excellent investigations of a group of English research workers^{17,18} how the mechanical co-operation of actin and L-myosin in the fibril takes place. It is shown by both electron micrographs and x-ray diagrams taken of the fibril during rest and contraction as well as by chemical research and interference micrographs^{19,20,21} that the so-called I-band of the fibril is built up of actin filaments aligned in parallel, which on one side are attached to the Z disc, and on the other slightly overlap the filaments lying in the so-called A-band. The latter is filled mainly with filaments of L-myosin oriented in parallel among the interstices of which the overlapping portion of the actin filaments is found (Fig. 2-12). During contraction the actin and L-myosin filaments shift in such a way that the actin filaments are drawn more deeply into the A-band until the I-band has disappeared. This is the mechanical process that brings about shortening.

It is easy to find a plausible series of chemical reactions by means of which the energy set free by the ATP splitting is used for a sliding movement of the actin filaments alongside the L-myosin filaments. Such a theory has been put forward²². I am not going to discuss it, because I want to confine myself to facts.

CONCLUSIONS

The findings I have discussed lead to the following conclusions:

1. The protein of the structures that produce every kind of muscular contraction and many kinds of cellular contraction splits ATP

2. The conditions leading to ATP splitting by means of this protein complex are identical for contractile structures of muscles and cells

3. On the other hand, the conditions that render these contractile ATPases active are different from the conditions that activate the other ATPases contained in muscles and cells: mersalyl concentrations that poison the activity of the contractile ATPases of muscles and cells completely and reversibly affect the other ATPases only slightly

4. If the ATP splitting induced by the contractile structures is inhibited, relaxation ensues from every kind of muscular contraction and many kinds of cellular contraction when ATP is present

5. If ATP is absent, all muscles and muscle-like cell structures become rigid

6. Consequently, ATP represents a plasticiser for the contractile structures of all muscles and many kinds of cells, if it is bound without being split, but an agent producing contraction, if it is split

7. In the skeletal muscle the contractile structures do not shorten because the individual protein filaments shorten by means of the ATP splitting. The contractile structures shorten because the filaments of two different proteins, the actin and the L-myosin, slide alongside each other. This knowledge means the end of all elastic theories of muscular contraction prevailing for the past hundred years.

8. Since the principle of interaction between ATP and the contractile structure is identical for every kind of muscular movement and for many kinds of cell movements, it must be assumed that not only the shortening of the skeletal muscle but also the contraction of smooth muscles and many kinds of cell contraction are produced by a sliding

of filaments. Moreover, such a mechanism enables us to understand why the contractile structures from the sarcoma cell to the mammalian muscles are invariably composed of two proteins.

BIBLIOGRAPHY

1. WEBER, H. H., and HOFFMANN-BERLING, H.: FILM Movements of Extracted Cells and Muscle Fibrils (Models) Produced by Adenosine Triphosphate (ATP). Hochschulfilm C 685/1954. Institut f. wissenschaftlichen Film, Göttingen, 1955
- 2a. HOFFMANN-BERLING, H.: Die Wasser-Glycerin-extrahierte Zelle als Modell der Zellmotilität, *Biochem et Biophys Acta* 10 628, 1953
- 2b. HOFFMANN-BERLING, H., and WEBER, H. H.: Vergleich der Motilität von Zellmodellen und Muskelmodellen, *Biochim et Biophys Acta* 10 629, 1953
3. VARGA, L.: Observations on the glycerol-extracted musculus psoas of the rabbit, *Enzymologia* 14 196, 1950
4. HOFFMANN-BERLING, H.: Adenosintriphosphat als Betriebsstoff von Zellbewegungen, *Biochim et Biophys. Acta* 14 182, 1954
5. HOFFMANN-BERLING, H.: Die glycerin-wassereextrahierte Telophasezelle als Modell der Zytokinese, *Biochim et Biophys Acta* 15 332, 1954
- 6a. WEBER, H. H., and PORTZEHL, H.: Kontraktion, ATP-cyclyus und fibrillare Proteine des Muskels, *Ergeb Physiol. biol. Chem., u. exp. Pharmacol.* 47 369, 1952
- 6b. WEBER, H. H., and PORTZEHL, H.: Transference of the muscle energy in the contraction cycle, *Progr. Biophys. and Biophys. Chem.* 4 60, 1954
7. PORTZEHL, H.: Gemeinsame Eigenschaften von Zell- und Muskelkontraktilität, *Biochim et Biophys. Acta* 14 195, 1954
- 8a. KUSCHINSKY, G., and TURBA, F.: Ueber die Bedeutung von Sulfhydrylgruppen für Prozesse am Aktomyosin, *Naturwissenschaften* 37 425, 1950.
- 8b. KUSCHINSKY, G., and TURBA, F.: Ueber den Chemismus von Zustandsänderungen des Aktomyosins, *Experientia* 6-103, 1950
9. HOFFMANN-BERLING, H.: Das Kontraktile Eiweiss undifferenzierter Zellen, *Biochim et Biophys Acta* 19 453, 1956
10. WEBER, H. H.: The link between metab-

- olism and motility of cells and muscles, *Symposia, Soc. Exp. Biol.* 9 271, 1955.
- 11 KUSCHINSKY, G., and TURBA, F. Ueber die Rolle der SH-Gruppen bei Vorgängen am Aktomyosin, Myosin und Atkin, *Biochim et Biophys. Acta* 6-426, 1951
 - 12 FORTZELL, H. Die Bindung des Erschlaffungsfaktors von Marsh an die Muskelgrana, *Biochim. et Biophys. Acta* 26 373, 1957
 - 13 HASSELBACH, W., and WEBER, H. H. Der Einfluss des MB-Faktors auf die Kontraktion des Fasermodells, *Biochim et Biophys. Acta* 11 160, 1953
 - 14 WEBER, H. H. Adenosine triphosphate and motility of living systems, *Harley Lectures* 49 37, 1953-1954.
 - 15 ULBRECHT, G., and ULBRECHT, M. Verkürzungsgeschwindigkeit und der Nutzeffekt der ATP-Spaltung Während der Kontraktion des Fasermodells, *Biochim et Biophys. Acta* 13 319, 1954
 - 16 HOFFMANN-BERLING, H. Der Mechanismus eines Neuen, von der Muskelkontraktion verschiedenen Kontraktionszyklus, *Biochim et Biophys. Acta* 27 247, 1958
 - 17a HUXLEY, A. F., and NIEDERGERGERKE, R. Structural changes in muscle during contraction interference microscopy of living muscle fibres, *Nature* 173-971, 1954
 - 17b HUXLEY, A. F. Present knowledge of the structure of striated muscle and its relation to function, *XX Internat'l Physiol. Congr.*, p. 219 Brussels, 1956 (Abstr. Reviews)
 - 18 HANSON, J., and HUXLEY, H. E. Structural basis of contraction in striated muscle, *Symposia, Soc. Exp. Biol.* 9 228, 1955
 - 19 HUXLEY, H. E., and HANSON, J. Quantitative studies on the structure of cross-striated myofibrils investigation by interference microscopy, *Biochim et Biophys. Acta* 23 229, 1957
 - 20 HANSON, J., and HUXLEY, H. E. Quantitative studies on the structure of cross-striated myofibrils investigations by biochemical techniques, *Biochim. et Biophys. Acta* 23 250, 1957
 - 21 HASSELBACH, W. Elektronenmikroskopische Untersuchungen an Muskelfibrillen bei totaler und partieller Extraktion des L-Myosins, *Z. Naturforsch.* 8b-449, 1953
 - 22 WEBER, H. H. Die molekularen Vorgänge bei der tierischen Bewegung, *Verhandl. deut. Orthop. Ges.* 44 13, 1956 (Beilageheft d. *Ztschr. f. Orthop.* 88)

CHAPTER 3 HEMODYNAMIC DETERMINANTS OF THE OXYGEN CONSUMPTION OF THE HEART WITH SPECIAL REFERENCE TO THE TENSION-TIME INDEX

*S. J. Sarnoff, E. Braunwald, G. H. Welch,
W. N. Stainsby, R. B. Case, and R. Macruz*

This investigation was conducted to acquire a detailed and precise appreciation of the influence of each of the various hemodynamic phenomena, including the tension-time index, on the nonfailing heart's utilization of oxygen. This required a heart preparation permitting (1) independent control of these phenomena, (2) essentially stable and nonfailing performance characteristics of the heart, (3) the ability to make determinations of total myocardial oxygen consumption with a high degree of precision.

The present understanding of myocardial function has been made possible to a substantial extent by study of the classical isolated heart or heart-lung preparations. The main virtue of these preparations is the ability to impose a degree of control on the experimental study that has not been readily achieved in the intact animal.

These palpable advantages are partially offset, however, by several serious drawbacks. The maximum aortic pressures and outputs obtained in such preparations are well below those that the heart of the intact animal can readily attain. The progressive deterioration in the performance characteristics of these hearts frequently complicates the interpretation of sequentially obtained data. Even recent workers feel it advisable to confine their observations to a period of 90 minutes. Finally, these hearts exhibit not only depressed Starling and ventricular function curves but also show a descending limb which, it is now known, the nonfailing heart does not present.¹ Because of these and other limitations it is generally agreed that isolated hearts are in incipient or overt failure.^{2,3}

An explanation was sought for the limited performance characteristics described above

in order to develop a preparation in which these disadvantages could be overcome while retaining the experimental advantages of isolation. It was reasoned that isolated hearts are deprived of their normal metabolic support and that their performance limitations might be explicable on this basis, i.e., the progressive deterioration of isolated preparations might be accounted for by the progressive deviation from normalcy of the blood supplying these hearts. An attempt was therefore made to maintain the biochemical environment of an isolated heart preparation at or near normal. In order to measure myocardial oxygen consumption (QO_2) more precisely, it was also thought desirable so to arrange the preparation as to permit sampling of completely mixed coronary venous blood

METHOD

The isolated supported heart (ISH) preparation used in these experiments is shown in Figure 3-1, its stability is shown in Figure 3-2, and evidence concerning its nonfailing performance characteristics shown in Figure 3-3. The techniques of measurement employed have been described in detail elsewhere.⁴ The preparation consists of an isolated dog heart, the left ventricle of which ejects blood through a Starling resistance and turbine flowmeter into a reservoir from which blood returns to the left atrium through a second, adjustable resistance. This constitutes a closed circuit except for the escape of coronary blood flow into the right heart, which receives no other blood. This completely mixed coronary venous blood is ejected through the pulmonary artery, its flow is

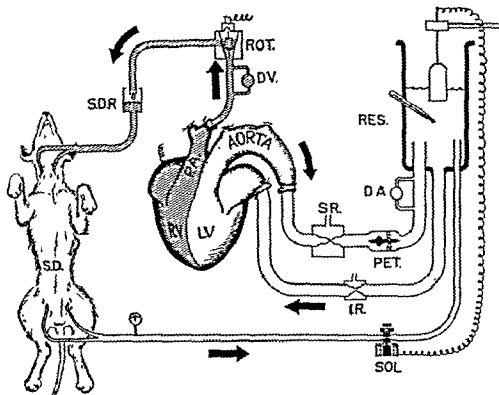


Fig 3-1 Schematic diagram of the ISH preparation. *SR* = air-filled Starling resistance. *PET* = Potter electro-turbine meter. *DA* = arterial densitometer. *RES* = reservoir. *IR* = water-filled inflow Starling resistance. *DV* = venous densitometer. *ROT* = rotameter. *SDR* = support dog reservoir. *SD* = support dog. *SOL* = solenoid valve electrically operated by microswitch at top of reservoir float.

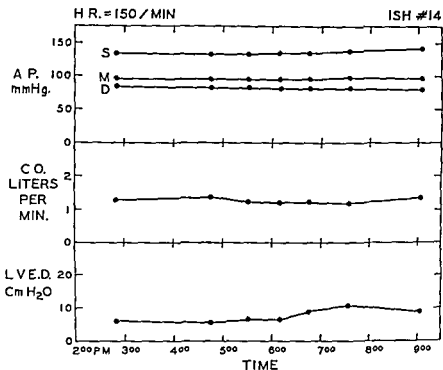


FIG 3-2 Aortic pressure, cardiac output, and left ventricular end-diastolic pressure measurements over a period of 62 hours in an ISH preparation.

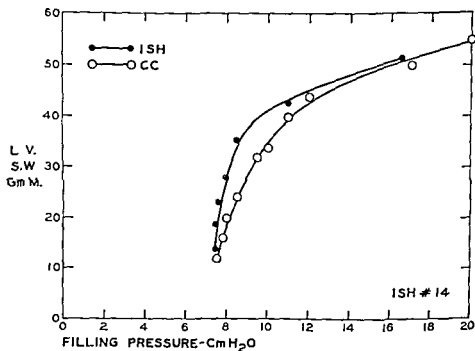


FIG. 3-3. A representative ventricular function curve from an open-chest dog with a complete circulation is represented by the open circles. Dog weight = 290 kg Heart weight = 174 gm Heart rate = 162/min. A representative ventricular function curve obtained from an ISH preparation is shown by the dots. Dog weight = 27.2 kg Heart weight = 204 gm. Heart rate = 150/min.

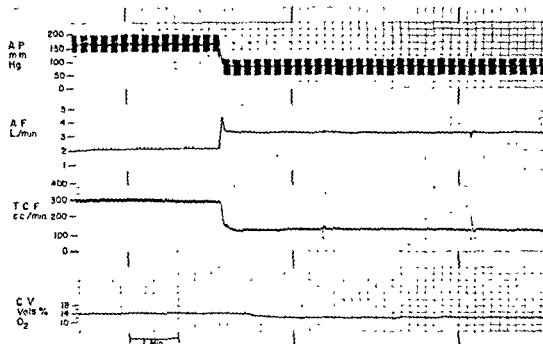


FIG. 3-4 AP = aortic pressure (full pulse and mean) AF = aortic flow TCF = coronary outflow CV = coronary venous blood oxygen content derived from the intraexperimentally determined calibration of densitometer $AF + TCF$ = cardiac output Outflow resistance abruptly lowered at point where AP falls and AF rises O_2 consumption at left = 23.2 cc/min O_2 consumption at right = 13.0 cc/min Lag period between change in coronary flow and change in coronary venous O_2 content is attributable to dead space, e.g., blood volume of coronary veins, right atrium and ventricle, pulmonary artery, and inflow tubing from pulmonary artery to cuvette of densitometer. Note the promptness with which the new steady state is achieved

metered, and delivered to the jugular veins of a second dog. Biochemically normal arterial blood from this support dog is simultaneously returned to the reservoir system at the same rate at which it leaves. Aortic pressures, cardiac output, and heart rate are independently controlled by the appropriate adjustments of the outflow resistance, the inflow resistance, and an electric pacemaker.

The O_2 and CO_2 contents of arterial and mixed coronary venous blood were determined gasometrically by the technique of Van Slyke and Neill. Coronary venous and, in some experiments, arterial blood saturations were also continuously recorded with densitometers⁵. In conjunction with continuous coronary flow measurement, it was possible to be certain of the presence of the equilibrium state (Fig. 3-4) at the time of sampling, and thus permit the application of

the Fick principle. Myocardial QO_2 was calculated as the product of coronary flow and the coronary arteriovenous O_2 difference. Unless otherwise noted, left ventricular minute work in kilogram meters was calculated as the product of cardiac output and the planimetrically integrated mean systolic aortic pressure in cm H_2O , obtained from 100 mm/sec pressure tracings.

External efficiency* was calculated as the ratio of minute work in kilograms to the product of minute QO_2 in cubic centimeters and the conversion factor of 2.06. The tension-time index (TTI) per beat in mm Hg seconds was obtained from the area under the systolic portion of the aortic pressure curve and is equal to the mean systolic pressure times the duration of systole. The TTI

* See Discussion p. 31

per minute is the product of T.T.I. per beat and the heart rate

The assumption was made that the $\dot{Q}O_2$ of the atria, and of the right ventricle which ejects only coronary flow, constitutes a relatively small portion of the total $\dot{Q}O_2$ and, more important, that their $\dot{Q}O_2$ does not change significantly in the course of a run in which left ventricular activity is varied. With this in view, the changes in heart $\dot{Q}O_2$ are attributed to changes in left ventricular activity alone.

The dogs from which the hearts for isolation were obtained weighed from 19.4 to 34.0 kg. The isolated hearts weighed from 178 to 352 gm. The left ventricular weights varied from 100 to 229 gm.

RESULTS

The results of 27 attempted experiments, of which 20 yielded fruitful data, will be presented in two sections.

Section I

The first group of experiments was devoted to examining the effects on myocardial $\dot{Q}O_2$ of the independent variation of cardiac output, mean aortic pressure and heart rate. Figure 3-5 shows the results of a representative experiment of the type in which (1) left ventricular work was elevated by raising aortic pressure while cardiac output and heart rate were held constant (pressure run) and (2) left ventricular work was elevated by increasing cardiac output while mean aortic

ISH #10

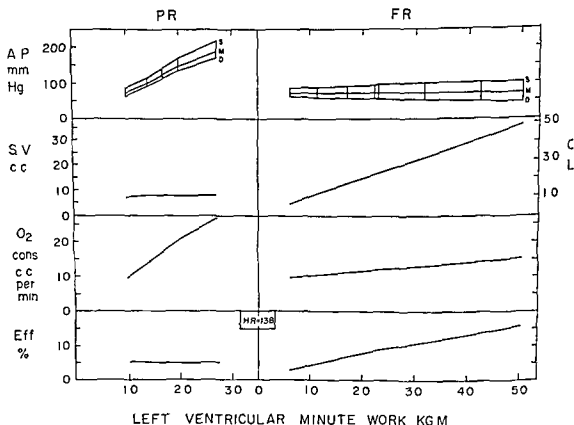


FIG 3-5. Contrasting effects on myocardial $\dot{Q}O_2$ of increasing work by increasing mean aortic pressure (pressure run = P.R.) and increasing work by increasing cardiac output (flow run = F.R.) Heart rate was held constant at 138/min. throughout. A.P. = aortic pressure. S.V. = stroke volume. Eff. = per cent efficiency. Left ventricular work in this experiment was calculated using mean aortic pressure.

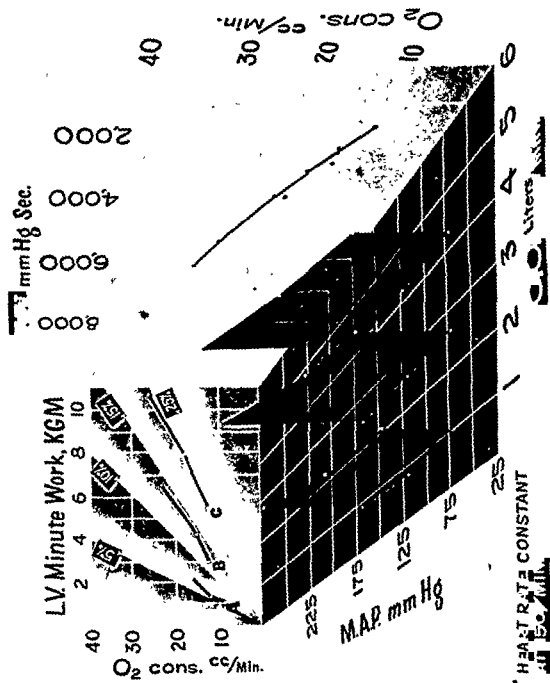


FIG. 3-6A The base panel shows the experimental conditions (cardiac output, aortic pressure, heart rate) when each determination of QO_2 was made. The height above the base panel of each experimental point represents its QO_2 . The rear panel shows the plot of left ventricular minute work in kilograms against myocardial QO_2 . The shaded lines on the rear panel labeled with percentage figures are (external) iso-efficiency lines. The right-hand panel shows the relationship between the Tension-Time Index (TTI) in mm Hg seconds and myocardial QO_2 in cc/min. Three pressure runs at low, medium, and high cardiac outputs. Note the negligible change in external efficiency as aortic pressure is increased within any given run.

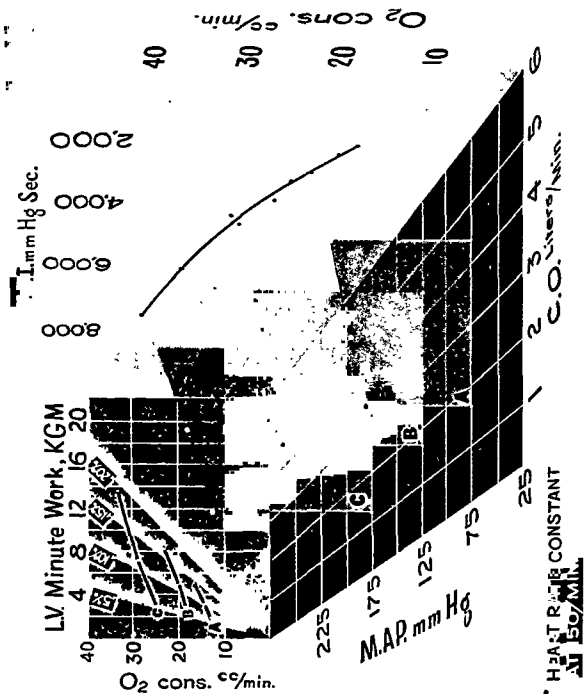


FIG 3-6B Three flow run at low, medium, and high mean aortic pressures. Note the increase in external efficiency within the course of any given flow run.

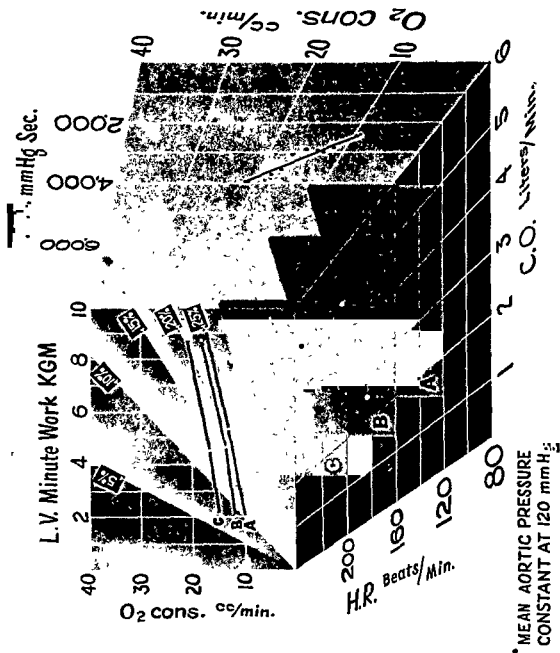


FIG. 3-6C Three flow runs at heart rates of 120, 160, and 200/min. Note the increased QO_2 at any given level of left ventricular work at the higher heart rates.

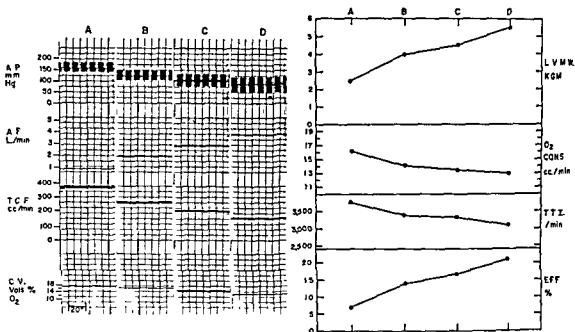


Fig. 3-7 Experiment which shows an increase in left ventricular minute work (*L.V.M.W.*) accompanied by a decrease in myocardial $\dot{Q}O_2$. Note parallelism between myocardial $\dot{Q}O_2$ and the Tension-Time Index (*TTI*). *A.F.* = aortic flow. *T.C.F.* = coronary flow. Sum of *A.F.* and *T.C.F.* = cardiac output. *C.V.* = coronary sinus blood O_2 content in volumes per cent as determined by intraexperimentally calibrated densitometer.

pressure and heart rate were held constant (flow run). In the pressure run, the increase in myocardial $\dot{Q}O_2$ paralleled the rise in aortic pressure and external efficiency therefore remained unchanged. Contrariwise, in the flow run the increase in work was accompanied by a relatively slight increase in $\dot{Q}O_2$ and a striking increase in external myocardial efficiency therefore occurred. In the pressure run, a 175 per cent work increase was accompanied by a 178 per cent increase on $\dot{Q}O_2$. In the flow run a 696 per cent work increase was accompanied by only a 53 per cent increase in $\dot{Q}O_2$. It should be further noted that during the flow run, in which mean aortic pressure was held constant while increasing stroke volume, a small increase in systolic aortic pressure did occur. The increment in myocardial $\dot{Q}O_2$ that did occur in the course of any given flow run was a function of the associated increment in aortic systolic pressure. This difference in the effects of pressure and flow on myocardial $\dot{Q}O_2$ was consistently observed in the twelve dogs in which these relationships were examined. Further data were then obtained to ascertain

whether the results shown in Figure 3-5 are limited to a narrow hemodynamic range or apply generally to wide ranges of pressures and flows. These results are shown in Figures 3-6A, 3-6B, and 3-6C. Figure 3-6A shows representative results from that type of experiment in which data from three successive pressure runs were obtained at each of three different cardiac output levels. It will be observed that, when aortic pressure alone was increased, the above-described parallel relationship between ventricular work and myocardial $\dot{Q}O_2$ obtained over the entire range of cardiac outputs examined.

Representative results from that type of experiment in which data from three successive flow runs were obtained at each of three different mean aortic pressure levels are shown in Figure 3-6B. It will be noted that the above-described non-parallel relationship between ventricular work and myocardial $\dot{Q}O_2$, when cardiac output alone is increased, obtains over the entire range of mean aortic pressures examined. That is, the increase in $\dot{Q}O_2$ was small relative to the large increments in ventricular work which occurred.

The influence of heart rate is seen in Figure 3-6C. With mean aortic pressure held constant at 120 mm Hg, and heart rate at 120/min, cardiac output was progressively increased from 1.2 to 4.9 liters/min. This was then repeated at heart rates of 160/min and again at 200/min. It will be observed that a higher heart rate at any given work level is accompanied by an increased myocardial

QO_2 and external myocardial efficiency is thereby decreased.

The above experiments strongly suggested that myocardial QO_2 bears little relation to the heart's external work per se. The absence of such an interrelationship could be even more convincingly demonstrated by the type of experiment shown in Figure 3-7. At a constant heart rate left ventricular minute work

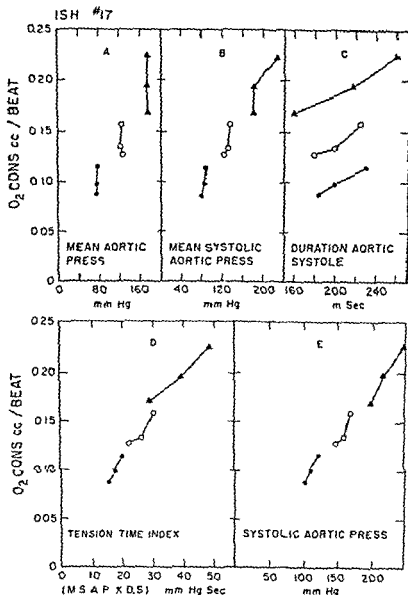


FIG 3-8 Relationships between certain hemodynamic parameters and myocardial QO_2 per beat (same experiment as in Figure 3-6B). Heart rate constant at 150/min throughout. Data from each of three flow runs are shown by the solid dots (at the low mean aortic pressure), open circles (at a medium mean aortic pressure), and triangles (at a high mean aortic pressure).

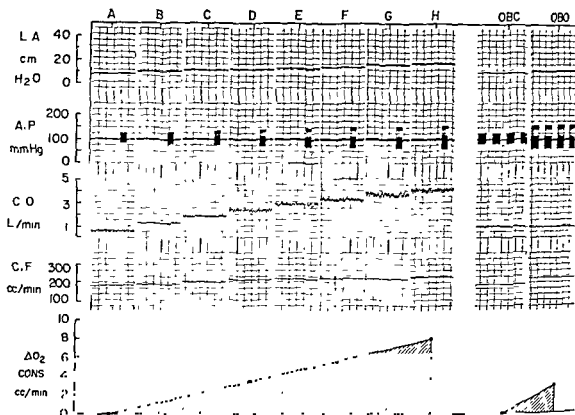


FIG 3-9 Points A through H show the hemodynamic changes and simultaneous $\dot{Q}O_2$ increment in the course of a flow run. Note the increase in peak systolic pressure as cardiac output was increased at constant mean aortic pressure. Shown on the right is the smaller $\dot{Q}O_2$ increment that resulted when the same change in peak systolic pressure was obtained after connecting a resonance chamber to the aortic line but, in this instance, without an increase in stroke volume (OBC to OBO). Heart rate constant throughout.

was progressively increased by increasing cardiac output while lowering aortic pressure to a lesser extent. The 128 per cent increase in work was accompanied by a 20 per cent decrease in $\dot{Q}O_2$.

Section II

When it became clear that the aortic pressure, or the development of tension by the heart, was the dominant influence in determining its O_2 utilization, further analysis and experimentation was designed for the purpose of exposing more precisely that particular pressure parameter which most closely relates to $\dot{Q}O_2$.

It was noted above (Fig 3-5) that, as might be anticipated, aortic systolic pressure rises when stroke volume is increased at any given mean aortic pressure and heart rate (flow run). This gave rise to the question of

whether all or any portion of that increment in $\dot{Q}O_2$ that does occur during a flow run is attributable to the increased systolic tension rather than to the increased fiber shortening (stroke volume) that takes place.

It can be seen from Figure 3-8 and Figures 3-6B and 3-6C that the $\dot{Q}O_2$ varied within the course of any given flow run in which mean aortic pressure was held constant. Also, while the mean systolic pressure did rise slightly during the course of a flow run (Fig. 3-8), it likewise did not correlate well with the $\dot{Q}O_2$ per beat; the $\dot{Q}O_2$ per beat changed substantially with only slight changes in mean systolic pressure. It will be further observed in Figure 3-8 that the duration of the tension state increased within the course of any given flow run. The product of the mean systolic pressure and the duration of systole yields the tension-time index in mm Hg seconds

(T.T.I.), which is an index of the total tension developed by the myocardium per beat. Figure 3-8 shows a good correlation between the T.T.I. and the $\dot{Q}O_2$ per beat

However, an equally good correlation between $\dot{Q}O_2$ per beat and peak systolic aortic pressure (Fig 3-8) was also consistently observed since, in the experiments described thus far, during the course of a flow run, the magnitude of the rise in peak aortic systolic pressure was similar to the rise of T.T.I. Thus, to ascertain whether the peak pressure developed or the T.T.I. is the responsible determinant of the heart's $\dot{Q}O_2$, two addi-

tional differentiating types of experiments were performed

The first type consisted of placing a resonance chamber into the aortic line. This made it possible to change peak systolic pressure without changing stroke volume, mean aortic pressure, or heart rate. A typical flow run (without the resonance chamber) is shown under *A* through *H* of Figure 3-9. With mean aortic pressure and heart rate held constant, cardiac output was progressively elevated and an increase in peak systolic pressure occurred. A similar increase in peak systolic pressure was then brought

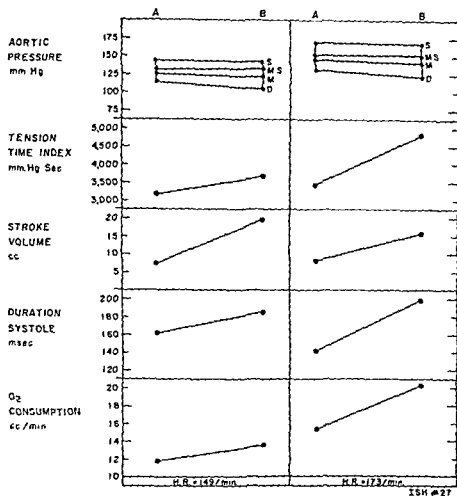


Fig. 3-10. Two experiments in one dog in which the T.T.I. was increased by increasing stroke volume and thus the duration of systole while decreasing aortic resistance so as to keep peak systolic pressure constant. Note the rise in myocardial $\dot{Q}O_2$ and T.T.I. S. = systolic, MS = mean systolic, M = mean, and D. = diastolic aortic pressures.

about by the resonance chamber without altering stroke volume and therefore without the concomitant increase in the duration of systole (*OBC* to *OBO* in Figure 3-9). In the latter instance a substantially smaller $\dot{Q}O_2$ increment was observed than during the prior flow run, indicating that the peak systolic pressure, per se, cannot be considered the fundamental determinant of $\dot{Q}O_2$.

In the second type of differentiating experiment, the peak systolic pressure was held constant, or allowed to fall slightly, while stroke volume was increased, in order to increase the duration of systole⁶ and thereby increase the T.T.I. The data from two such experiments are shown in Figure 3-10. It will be observed that whereas peak systolic pressure was held constant, the T.T.I. rose as a result of an increasing stroke volume. A rise in $\dot{Q}O_2$ accompanied this observed rise of the T.T.I.

The relationship between myocardial $\dot{Q}O_2$ and the T.T.I. showed a convincing correlation in 35 of the 37 experiments in the twelve dogs in which the T.T.I. was determined.

DISCUSSION

Previous investigators who have studied the effect of varying the hemodynamic parameters on myocardial $\dot{Q}O_2$ in isolated heart or heart-lung preparations of the dog, cat, tortoise, and frog emphasized various hemodynamic determinants of myocardial $\dot{Q}O_2$. It has been observed that increasing the heart rate increases the $\dot{Q}O_2$,^{7,8} and the above data confirm this. It has also been held that the energy of contraction is directly related to end-diastolic ventricular fiber length without regard to the nature of the contraction.^{9,10,11,12} A correlation between $\dot{Q}O_2$ and external work was found by some observers¹³ but in these investigations there was little or no separation of the effects of pressure and flow on the $\dot{Q}O_2$. Many have observed that a given increase in the work of the heart produced by raising aortic pressure while maintaining cardiac output constant resulted in a higher $\dot{Q}O_2$ than when a similar increase in work was achieved by raising cardiac output at a constant aortic pressure.^{12,14,15,16,17,18,19,20}

The data presented above in Section I are also consonant with the observations of this latter group, as well as with the early observations of Rohde,²¹ who concluded that the heart's $\dot{Q}O_2$ is proportional to the product of ventricular pulse pressure and heart rate. It is generally agreed, however, that the isolated heart or heart-lung preparations studied in all the above investigations were either in incipient or varying degrees of overt failure. Since the preparation used in these experiments can be considered essentially nonfailing,⁴ it is now clear that these observations apply to the nonfailing heart as well. Biochemical evidence supporting this position is present in the experiments of Nachmannsohn²² on skeletal muscle wherein phosphocreatine breakdown was observed to be greater during isometric than during isotonic contraction. Recently, preliminary observations by Wollenberger²³ suggest that the myocardium also expends phosphate bond energy chiefly in developing tension rather than in emptying.

The discrepancy between the relative O_2 costs of pressure and flow has frequently been observed in the isolated heart or heart lung as noted above, as well as in the dog with a complete circulation.²⁴ However, that increase of myocardial $\dot{Q}O_2$ which does occur at any mean aortic pressure with an increased cardiac output may now reasonably be attributed to the increased total tension (T.T.I.) resulting from the increased mean systolic pressure and increased duration of the tension state. This latter view is supported by the data in Section II. The right-hand panels of Figure 3-6, as well as Figures 3-7, 3-8, and 3-10 demonstrate the variety of circumstances under which observations were made and indicate that whichever hemodynamic parameter was varied, the relationship between T.T.I. and myocardial $\dot{Q}O_2$ obtained. *It appears, therefore, that, in any given functional state of the beating heart, the T.T.I. (mean systolic pressure times duration of systole) is the principal, if not the sole, determinant of myocardial O_2 utilization. If the correlations observed above are causal rather than coincidental, then the above data may*

result in the derivation of a more meaningful relationship between the O_2 used by contracting heart muscle and the physiologic purpose for which it is used.

At any given filling pressure, the myocardial QO_2 can vary over a substantial range and will be a function of the TTI developed during the course of the contraction²⁴. It appears, therefore, that the heart's QO_2 is determined not solely by the filling pressure prior to contraction, but rather by the events occurring subsequent to the onset of contraction from any given filling pressure. If marked changes in ventricular distensibility do not occur, this view could imply that similar considerations apply to the relationship between fiber length and myocardial QO_2 . The validity of this latter position, however, still awaits satisfactory experimental demonstration.

A variable not found possible to control in the above described experiments was the rate of the development of systolic tension while holding the other variables constant. The possibility must be considered, therefore, that these experiments are incomplete in that it has not been determined whether the rate of development of tension modifies the relationship between QO_2 and TTI.

The term *external efficiency* as used herein refers to the ratio of external work to QO_2 and conforms to the general usage of the term *efficiency* as applied to the heart. It is suggested herewith that the term *internal efficiency* refer to the ratio between the *actual total tension* developed by the myocardium and its QO_2 . *External efficiency* is primarily meaningful in terms of the relationship between the heart and the total organism but is relatively uninformative about the energetics of contracting myocardium. In contrast, the term *internal efficiency* is designed to expose the meaningful relationship between that aspect of the heart's contraction which requires O_2 (total developed tension) and the amount of O_2 consumed. In the absence of any ready means of measuring the *actual total tension* developed, the area beneath the systolic portion of the pressure curve (TTI in mm Hg seconds) has been

used as an *index* of the actual total myocardial fiber tension. The ratio of TTI to QO_2 thus provides an *internal efficiency index*. Any discrepancy between *internal efficiency* and the *internal efficiency index* will revolve around the extent to which ventricular radius changes occur and considerations involving Laplace's law* therefore apply, for it is clear from such considerations that the same intraventricular TTI will require a greater myocardial fiber tension in a large heart than in a small one. If it is assumed that the basic relationship is between the total actual tension developed by the myocardium rather than the reflection thereof in the observed TTI, then both the low internal and external efficiency of the greatly dilated or failing heart can logically be attributed, at least in part, to the interrelationship between Laplace's law and the dependence of the heart's QO_2 on its actual total developed tension. For example, if the dilated heart is called upon to develop any given intraventricular pressure, the myocardial fiber tension will be greater and its QO_2 will thus also be greater than that of the small heart producing the same intraventricular pressure at the same stroke volume and heart rate. In this sense the dilating heart may be thought of as one with an O_2 requirement that approaches or finally could exceed the limit of the O_2 available to it, especially when O_2 availability is limited by disease. It should be emphasized that such considerations are valid only if the assumption of the basic relationship between QO_2 and *actual total tension* developed is a correct one.

SUMMARY AND CONCLUSIONS

An isolated heart preparation was developed that was stable, nonfailing, and retained performance characteristics comparable to the heart of the open chest, anesthetized dog because of continued metabolic support derived from the blood of a second dog. This

* For a cylinder, $T = P \times R$ where T = wall tension, P = intraluminal pressure, R = radius. For further discussion see Rushmer²⁵ and Burton (p. 74).

preparation was employed in an examination of the hemodynamic determinants of myocardial oxygen consumption ($\dot{Q}O_2$). Precise control of cardiac output, aortic pressures, heart rate, and ventricular filling pressure was readily obtained.

Only under special circumstances was there any straightforward relationship between external work and $\dot{Q}O_2$. When left ventricular work was increased by elevating aortic pressure, while holding cardiac output and heart rate constant, $\dot{Q}O_2$ rose *pari passu* with aortic pressure and work, leaving external efficiency unchanged. In contrast, when work was increased manyfold by elevating cardiac output at a constant heart rate and mean aortic pressure, only a small increase in $\dot{Q}O_2$ occurred, thereby markedly increasing external efficiency. When external work was more than doubled by markedly elevating cardiac output while lowering aortic pressure to a lesser extent, myocardial $\dot{Q}O_2$ actually diminished. When heart rate was increased while holding cardiac output, mean aortic pressure, and therefore work constant, $\dot{Q}O_2$ increased, thereby lowering external efficiency.

It was further observed that when cardiac output was increased at a constant mean aortic pressure and heart rate, the relatively small increase in $\dot{Q}O_2$ that did occur was accompanied by an increase in aortic systolic pressure. This suggested the possibility that myocardial $\dot{Q}O_2$ is related solely or predominantly to the development of tension without regard to the amount of fiber shortening (stroke volume) that takes place. In experiments that were designed to ascertain whether any particular pressure parameter would consistently correlate with observed changes in myocardial $\dot{Q}O_2$, the aortic systolic pressure, mean systolic pressure, mean pressure, and diastolic pressure and also the total area under the systolic pressure curve (TTI) were examined. The $\dot{Q}O_2$ correlated only with the TTI (in 35 out of 37 experiments) regardless of the hemodynamic variations imposed. It was concluded that myocardial $\dot{Q}O_2$ is determined by the total tension developed by the myocardium insofar as this is expressed by the area beneath the systolic

pressure curve and that myocardial $\dot{Q}O_2$ depends primarily upon the sequence of events after the onset of contraction rather than on the filling pressure per se.

The role of Laplace's law in the interpretation of these data and the possible application of this information to the interpretation of the low external and internal efficiency of the failing heart was discussed.

ACKNOWLEDGMENTS

Acknowledgment is made to the *American Journal of Physiology*, in which the following papers have been published:

1 SARNOFF, S. J., CASE, R. B., WELCH, G. H., JR., BRAUNWALD, E., STAINSBY, W. N.: Performance characteristics and oxygen debt in a non-failing, metabolically supported isolated heart preparation, *Am. J. Physiol.* 192:141, 1958.

2 SARNOFF, S. J., BRAUNWALD, E., WELCH, G. H., JR., CASE, R. B., STAINSBY, W. N., MACRUZ, R.: Hemodynamic determinants of the oxygen consumption of the heart with special reference to the tension-time index. *Am J Physiol* 192:148, 1958.

BIBLIOGRAPHY

1. SARNOFF, S. J., and BERGLUND, E.: Ventricular function. Starling's law of the heart studied by means of simultaneous right and left ventricular function curves in the dog. *Circulation* 9:706, 1954.
2. LORBER, V.: Energy metabolism of the completely isolated mammalian heart in failure. *Circulation Res* 1:298, 1953.
3. KATZ, L. N., WISE, W., and JOCHIM, K.: The dynamics of the isolated heart and heart-lung preparations of the dog. *Am J Physiol* 143:463, 1945.
4. SARNOFF, S. J., CASE, R. B., WELCH, G. H., JR., BRAUNWALD, E., STAINSBY, W. N.: Performance characteristics and oxygen debt in a non-failing metabolically supported isolated heart preparation, *Am J. Physiol* 192:141, 1958.
5. SABISTON, D. C., JR., KHOURI, M., and GREGG, D. E.: Use and application of the cuvette densitometer as an oximeter. *Circulation Res.* 5:125, 1957.

- 6 BRAUNWALD, E., SARNOFF, S. J., and STAINSBY, W. N.: Determinants of duration and mean rate of ventricular ejection, *Circulation Res* 6:319, 1958.
- 7 EVANS, C. L.: The mechanism of cardiac acceleration by warmth and by adrenalin, *J Physiol* 51:91, 1917.
- 8 COHN, A. E., and STEELE, J. M.: The influence of frequency of contraction of the isolated mammalian heart upon the consumption of oxygen, *Am. J. Physiol.* 113: 654, 1935
- 9 STARLING, E. H., and VISSCHER, M. B.: The regulation of the energy output of the heart, *J. Physiol* 62:213, 1927.
- 10 STARLING, E. H.: *The Linacre Lecture on the Law of the Heart* Given at Cambridge, 1915 London, Longmans, Green & Co., 1918
- 11 HEMINGWAY, A., and FER, A. R.: The relationship between volume of the heart and its oxygen usage, *J. Physiol.* 63:299, 1927.
- 12 DECHERD, G., and VISSCHER, M. B.: The relative importance of the performance of work and the initial fiber length in determining the magnitude of energy liberation in the heart, *Am J Physiol* 103:400, 1933
- 13 BARCROFT, J., and DIXON, W. E.: The gaseous metabolism of the mammalian heart, *J Physiol* 35:182, 1907
- 14 STELLA, G.: The oxygen consumption of the tortoise heart, its dependence upon diastolic volume and on the mechanical conditions of systole, *J Physiol* 72:247, 1931
- 15 EVANS, C. L., and MATSUOKA, Y.: The effect of various mechanical conditions on the gaseous metabolism and efficiency of the mammalian heart, *J Physiol* 49:378, 1915
- 16 GOLLWITZER-MEIER, K.: Die Energetik des Säugetierherzens, *Klin. Wchnschr.* 18:225, 1939
- 17 GREMELS, H.: Zur Physiologie und Pharmakologie der Energetik des Säugetierherzens, *Arch. exper. Path. u. Pharmacol* 169:689, 1933.
- 18 KIESE, M., and GARAN, R. S.: Mechanische Leistung Grosse und Sauerstoffverbrauch des Warmblüterherzens, *Arch exper Path. u Pharmacol* 188:226, 1938.
- 19 KATZ, L. N., JOCHIM, K., LINDNER, E., and LANDOWNE, M.: The effect of varying resistance-load and input-load on the energetics of the surviving mammalian heart, *Am J Physiol* 134:636, 1941
- 20 ALLELA, A., WILLIAMS, F. L., BOLENE-WILLIAMS, C., and KATZ, L. N.: Interrelation between cardiac oxygen consumption and coronary blood flow, *Am J Physiol* 183:570, 1955
- 21 RONDE, E.: Über den Einfluss der mechanischen Bedingungen auf die Taugkeit und den Sauerstoffverbrauch der Warmblüterherzens, *Arch exper. Path. u. Pharmacol* 68:401, 1912.
- 22 NACHMANN-SOHN, D.: Über den Zerfall der Kreatinophosphorsäure im Zusammenhang mit der Tätigkeit des Muskels, *Biochem Ztschr.* 208:237, 1929
- 23 WOLLENBERGER, A.: Relation between work and labile phosphate content in the isolated dog heart, *Circulation Res* 5:175, 1957.
- 24 SARNOFF, S. J.: *Unpublished observations*
- 25 RUSHMER, R. F.: *Cardiac Diagnosis Physiologic Approach* Philadelphia, W. B. Saunders Co., 1955, pp 110, 126.

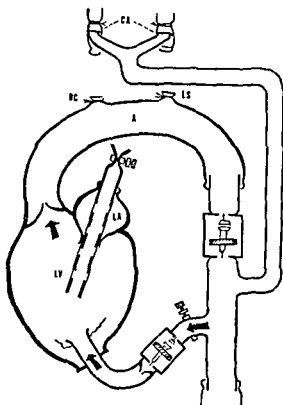


FIG 4-2. Schematic diagram showing preparation for producing aortico-left ventricular regurgitation, including check valve and flowmeter for the recording of regurgitant flow during diastole. Second flowmeter records total left ventricular output (minus coronary flow). A perforated tube placed in the left ventricle through the left atrial appendage and mitral valve to superimpose mitral regurgitation on metered aortic regurgitation. Effective cardiac output = total minus regurgitant flow. LA = left atrium, LV = left ventricle, A = aorta, BC = brachiocephalic artery, LS = left subclavian artery, CA = carotid arteries. Screw clamp on regurgitant tubing allows control of volume of regurgitation.

duced, as shown in Figure 4-2, by providing a pathway from the thoracic aorta to the prosthesis sutured into the left ventricular apex. This was done in eleven dogs weighing from 19.5 to 27.0 kg and averaging 24.7 kg. A check valve permitted reflux from aorta to left ventricle only during diastole. This regurgitant flow, which will be referred to hereinafter as aortic regurgitant flow, was metered by a Potter turbine flowmeter in the regurgitant pathway. This flow could be

regulated by an externally applied screw clamp. Total left ventricular output minus coronary flow (effective plus regurgitant flows) was also continuously metered with a second flowmeter as shown in Figure 4-2. In order to superimpose mitral regurgitation upon any given level of aortic regurgitation, a tygon tube with multiple perforations was inserted through the mitral valve by way of the left atrial appendage. This could be withdrawn and replaced at will. Effective cardiac output (systemic flow) was calculated as the difference between total aortic and regurgitant flows. Total left ventricular stroke work in gram-meters was calculated as the product of total left ventricular output in cubic centimeters and mean aortic pressure in centimeters H₂O divided by heart rate. Effective left ventricular stroke work in gram-meters was calculated as the product of effective systemic flow and mean aortic pressure in centimeters H₂O divided by heart rate. The aortic regurgitant gradient was estimated as the difference between mean aortic diastolic pressure, obtained by planimetry, and left ventricular end-diastolic pressure. The regurgitation index was calculated as the product of the duration of diastole and the aortic regurgitant gradient.

RESULTS

Mitral Regurgitation

HEMODYNAMIC EFFECTS OF GRADED MITRAL REGURGITATION The hemodynamic consequences of stepwise increases in mitral regurgitation are shown in Figure 4-3. Regurgitant flow was varied from zero to 4.24 liters per minute, a value more than twice the initial cardiac output present before mitral regurgitation was induced. Surprisingly modest changes in effective cardiac output, aortic pressure, and left atrial mean and "Z" point pressures occurred. Calculated peripheral resistance rose slightly. These data are representative of 28 similar experiments in eleven dogs.

THE EFFECT ON MITRAL REGURGITANT FLOW OF INCREASED RESISTANCE TO VENTRICULAR EJECTION. With any given mitral regurgitant

orifice, a stepwise increase in the resistance to left ventricular outflow produced approximately parallel increases in mitral regurgitant flow with a reciprocal fall of forward flow. The results of one such experiment are shown in Figure 4-3 and are representative of 23 of the 26 such experiments performed in eleven dogs.

THE INFLUENCE OF MITRAL REGURGITATION ON THE EFFECTIVE LEFT VENTRICULAR FUNCTION CURVE Results representative of the nine such experiments performed in six dogs

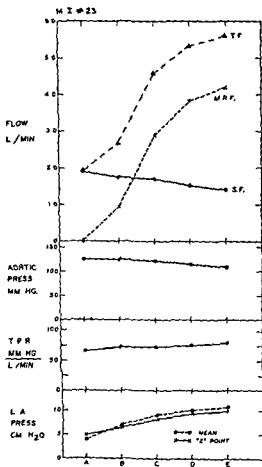


FIG 4-3 MI Exp 23 The hemodynamic effects of increasing ventriculoatrial regurgitation. TF = total flow. MRF = ventriculoatrial (mitral) regurgitant flow. SF = forward cardiac output (minus coronary flow). Other panels from above downward are mean aortic pressure, calculated total peripheral resistance (TPR), and left atrial mean and "Z" point pressures (LA). Dog weight = 25.9 kg.

are shown in Figure 4-5. In this experiment only a slight depression of the ventricular function curve was observed in the presence of these substantial mitral regurgitant flows (2.1 to 2.4 liters per minute). In one experiment, in which a series of ventricular function curves were obtained with serially increased mitral regurgitant volumes, progressive depressions of the ventricular function curve occurred.

THE RELATIVE INFLUENCE OF MITRAL REGURGITANT FLOW AND EFFECTIVE CARDIAC OUTPUT ON VENTRICULAR FILLING PRESSURE. The above data suggested that the filling pressure required to provide any given increment of effective cardiac output exceeded that which was associated with a similar increment of mitral regurgitant flow. The re-

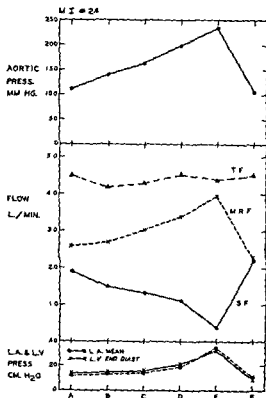


FIG 4-4 MI Exp 24 Points A through E show the effects of increasing the resistance to left ventricular ejection in the presence of a constant regurgitant orifice. Aortic resistance was returned to original level at point F. Symbols same as in Figure 4-3. Dog weight = 24.1 kg.

sults of an experiment designed to examine this hypothesis more critically are illustrated in Figure 4-6. By the appropriate adjustments of aortic resistance, mitral regurgitant orifice size, and blood volume, it was possible to decrease forward and increase regurgitant flows reciprocally, so as to maintain mean left atrial, left atrial "Z" point, aortic pressures, and heart rate constant, or almost so. Little change in filling pressure occurred while the increase in mitral regurgitant flow was 3.7 times the decrease in effective cardiac output. Total flow doubled. These results are representative of seven such experiments in five dogs.

INFLUENCES THAT MODIFY THE EFFECT OF MITRAL REGURGITATION ON LEFT ATRIAL PRESSURE The effect on left atrial "Z" point pressure of progressively increasing mitral regurgitant flow was examined at two different effective cardiac outputs (650 and 1750 cc/min) at comparable aortic pressures and

heart rates (solid curves in Figure 4-7). When effective cardiac output was maintained at a low level, left atrial pressure was 3.5 cm. H_2O before mitral regurgitation was induced; 4.74 liters/min of regurgitation raised left atrial pressure 4.5 cm. H_2O . In contrast, when effective cardiac output was maintained at a higher level and left atrial "Z" point pressure was 10 cm H_2O before mitral regurgitation was induced, 4.09 liters/min of regurgitation raised left atrial pressure 11.5 cm H_2O . It was consistently observed in the ten comparisons of this type performed in nine dogs that the higher the ventricular filling pressure prior to mitral regurgitation, the greater was the subsequent elevation of filling pressure produced by any given amount of regurgitation.

Figure 4-7 also confirms the data previously shown in Figure 4-6 in that a given increase in total flow produced by increasing effective cardiac output (broken lines) required much greater elevations in filling pressure than did similar increases in total flow produced by increasing mitral regurgitant flow (solid lines).

THE EFFECT ON LEFT ATRIAL PRESSURES OF PHARMACOLOGICALLY ELEVATING THE VEN-

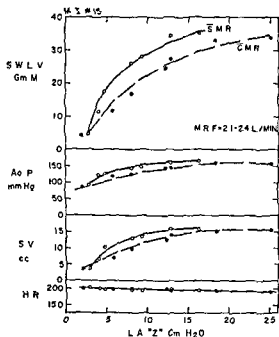


FIG 4-5 M.I. Exp 15. Effect on the left ventricular function curve of the presence of mitral regurgitant flow (MRF.) SWLV = effect left-ventricular stroke work in gram meters. Ao P = mean aortic pressure. SV = stroke volume. HR = heart rate/min. LA "Z" = left atrial "Z" point pressure. Dog weight = 25.4 kg.

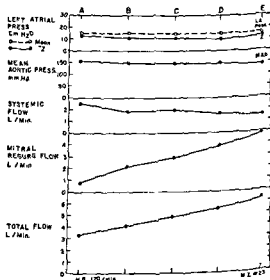


FIG 4-6 M.I. Exp 23. Effects of reciprocally varying forward and regurgitant flows at a constant left atrial pressure. HR. constant at 170/min. Dog weight = 25.9 kg.

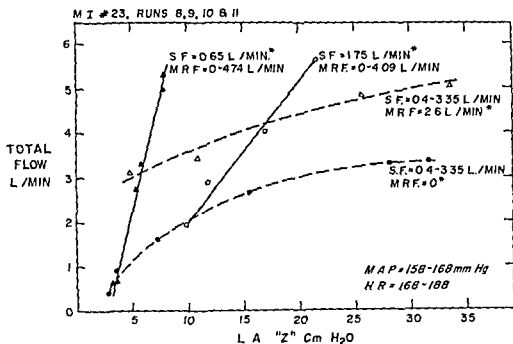


FIG 4-7 MI Exp 23 Solid lines show effect on left trial "Z" point pressure of increasing total flow by increasing regurgitant flow (MRF) while holding forward cardiac output (SF) constant Triangles shows change in LA "Z" point pressures while increasing regurgitant flow when forward flow was constant at 0.65 L/min Open circles show the effect of increasing regurgitant flow when forward flow was held constant at 1.75 liters/min Broken lines show the effect on LA "Z" point pressure of increasing total flow by increasing forward flow while regurgitant flow was held constant at zero (solid dots) and at 2.6 liters per minute (open triangles) Asterisk denotes value held constant during any given run MAP = mean aortic pressure and HR = range of heart rates throughout

TRIGULAR FUNCTION CURVE IN THE PRESENCE OF MITRAL REGURGITATION Figure 4-8 shows the results of one experiment in which, after mitral regurgitation had been produced, 0.4 mg/kg of mephentermine sulfate (Wyamine) was injected intravenously. The administration of this agent, which has been shown to elevate the ventricular function curve,* was followed by a substantial fall in mean left atrial and "Z" point pressures even though mitral regurgitant flow increased and aortic pressure and effective stroke volume were also elevated

Aortic Regurgitation

THE HEMODYNAMIC CONSEQUENCES OF GRADED INCREASES IN THE VOLUME OF AORTIC REGURGITATION The results of one of fourteen such experiments in nine dogs are shown

in Figure 4-9. As aortic regurgitant flow was increased, effective cardiac output decreased substantially. This was always accompanied by a widening of the pulse pressure and lower diastolic pressure. Mean pressure was usually, but not always, slightly diminished. Systolic pressure failed to rise in two of the experiments of this type, one of which is shown in Figure 4-9. Calculated total peripheral resistance rose markedly. Left ventricular end-diastolic pressure rose to high levels while mean left atrial pressure exhibited only a slight elevation. Thus in end diastole, wide discrepancies between left ventricular and left atrial pressures were generally observed.

THE EFFECT OF AORTIC REGURGITATION ON THE LEFT VENTRICULAR FUNCTION CURVE. Ventricular function curves were obtained by

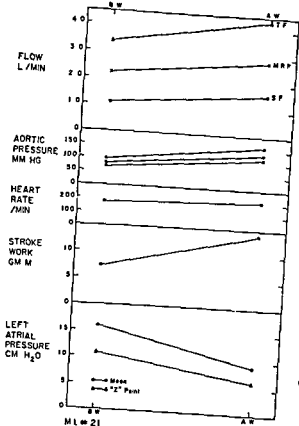


FIG. 4-8 MI Exp 21 Effects on L.A. mean and "Z" point pressures, forward flow, regurgitant flow, aortic pressure, stroke work, and heart rate following the administration of 0.4 mg/kg of mephentermine sulfate

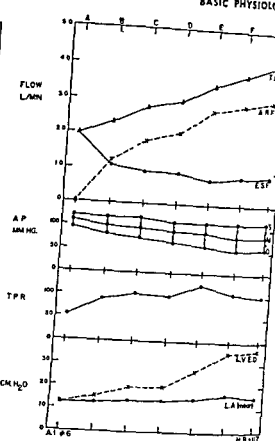


FIG. 4-9 AI Exp 6 Hemodynamic effects of graded increases in aortic regurgitant flow (ARF). T.F. = total flow, ESF = effective cardiac output, A.P. = aortic pressure, T.P.R. = calculated total peripheral resistance, L.A. = left atrial pressure, L.V.E.D. = left ventricular end-diastolic pressure Dog weight = 265 kg

stepwise blood infusion in nine experiments in seven dogs. After each infusion, observations were made with and without a constant amount of aortic regurgitant flow by alternately closing and opening the regurgitant pathway. Figure 4-10A shows the effective stroke work plotted against left ventricular end-diastolic pressure with and without aortic regurgitation. A marked depression of the effective left ventricular function curve was observed. Figure 4-10B shows the total left ventricular stroke work done at various filling pressures in the presence of aortic regurgitation, as well as its partition into effective stroke work and that component of the stroke work expended on the regurgitant stroke volume.

HEMODYNAMIC CONSEQUENCES OF CHANGING CARDIAC OUTPUT. Stepwise increases in

total cardiac output were brought about by serial blood infusions in one experiment and observations made on the parameters shown in Figure 4-11. It was observed that the higher the cardiac output the greater was the ratio between effective and total cardiac output. An increase in aortic regurgitant flow did occur, but this was slight even in the presence of an elevation of aortic pressure and an accompanying increase in the aortic regurgitant gradient. The observation that the increase in aortic regurgitant flow was only slight may be explained by the finding that, with the increase in total stroke volume, the diastolic period shortened³ and therefore aortic regurgitation index (the product of the aortic regurgitant gradient and the duration of diastole) rose only slightly. This view is

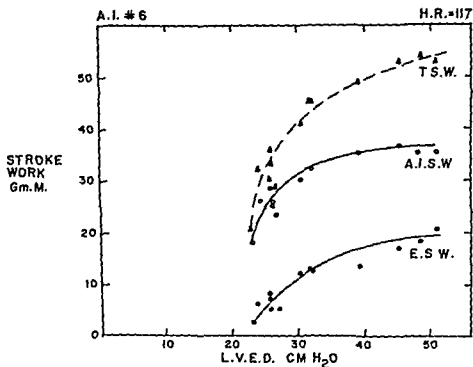
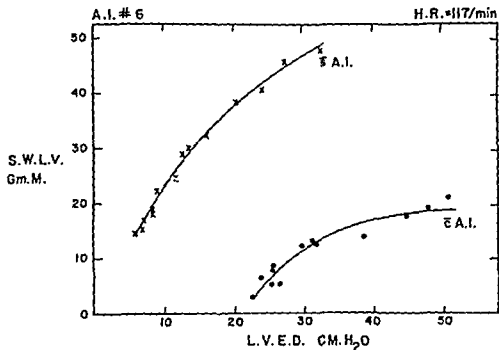


FIG 4-10 A1 Exp 6 (Upper) Effective left ventricular function curves without (\bar{x} A.I.) and with (\bar{A} I) an aortic regurgitant flow of 26 liters/min S.W.L.V. = left ventricular stroke work in gram meters, L.V.E.D. = left ventricular end-diastolic pressures (Lower) Plot showing the total left ventricular stroke (T.S.W.) and its partition into effective stroke work (E.S.W.) and the component of stroke work expended on the regurgitant stroke volume (A.I.S.W.). This plot was constructed from data obtained during the experiment with aortic regurgitation shown in A

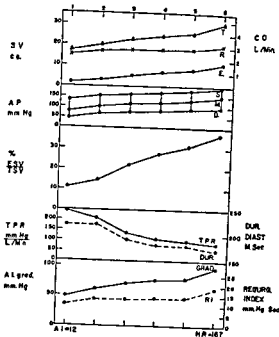


FIG 4-11. A.I. Exp 12 Hemodynamic effects of increasing cardiac output by blood infusion (points 1 through 6) with a constant aortic regurgitant orifice T = total flow, R = aortic regurgitant flow, E = effective cardiac output SI' = stroke volume, CO = cardiac output, AP = aortic pressure ESV/TSV = the ratio of effective to total stroke volume TPR = calculated total peripheral resistance DUR = duration of diastole in milliseconds $A1$ grad = aortic regurgitation gradient. RI = aortic regurgitation index HR = heart rate at 167/min constant throughout. Dog weight = 220 kg

supported by the parallelism seen in Figure 4-11 between changes in the calculated regurgitation index and observed changes in regurgitant flow. Further, the fall in calculated total peripheral resistance that accompanied the increase in effective cardiac output limited the rise in the aortic regurgitant gradient and thus the aortic regurgitation index. It appears, therefore, that the decreases in the duration of diastole and peripheral resistance account for the increased ratio between effective and total stroke volume when cardiac output is increased in the presence of aortic regurgitation.

THE EFFECTS OF MITRAL REGURGITATION SUPERIMPOSED ON AORTIC REGURGITATION

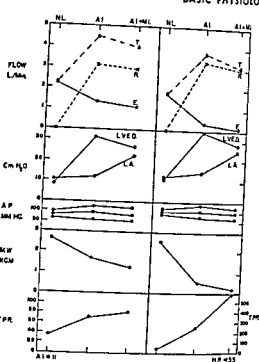


FIG 4-12 A.I. Exp. 11. Hemodynamic effects of superimposing mitral regurgitation on aortic regurgitation NL = no lesion, that is, prior to the induction of aortic regurgitation AI = after inducing aortic regurgitation $AI + MI$ = after mitral regurgitation was superimposed on the aortic regurgitation T = total flow.* R = aortic regurgitant flow, and E = effective flow in liters/minute. $LV ED$ = left ventricular end-diastolic pressure. LA = mean left atrial pressure. AP = aortic pressure MW = effective minute work in kilogram meters, TPR = calculated total peripheral resistance HR = heart rate per minute at 133 constant throughout. Dog weight = 250 kg. Note the fall in minute work paralleling the fall in $LV ED$ when mitral regurgitation was added.

Nine experiments were performed in four dogs in which the effect on circulatory dynamics of inducing aortic regurgitation were first observed. Mitral regurgitation was then superimposed by means shown in Figure 4-2. The results of two such experiments are shown in Figure 4-12. When aortic regurgitation was induced, the previously demonstrated hemodynamic changes occurred. When

* This is the sum of effective cardiac output and aortic regurgitant flow but does not include the unmeted mitral regurgitant flow.

mitral regurgitation was added, even though the aortic regurgitant flow was slightly diminished, effective cardiac output fell further from its already depressed level. Simultaneously, left ventricular end-diastolic pressure fell from its high level while mean left atrial pressure rose, thus diminishing the discrepancy that had been present between them when the mitral valve was competent. When mitral regurgitation was added, the fall in left ventricular end-diastolic pressure was accompanied, as might be anticipated, by a fall in the effective stroke work.

DISCUSSION

The application to disease states in man of data derived from acute canine experiments that simulate such states is necessarily limited. This is largely because of possible species differences and the lack of the gradual development of the lesion, together with whatever compensatory factors may come into play. Nevertheless, certain palpable advantages are obtained from the type of preparation herein described in which (1) valvular regurgitation is effectively produced, (2) the regurgitant flow is directly metered, and (3) various hemodynamic parameters, including the volume of regurgitant flow, can be independently controlled. A more complete analysis of the hemodynamic effects of changing one of the variables can thereby be obtained.

It was of considerable interest to observe the relatively small changes in forward cardiac output and aortic pressure when mitral regurgitation was induced. Further, these were accompanied by only small changes in mean left atrial and left ventricular filling pressures. Total cardiac output increased markedly, and little depression of the effective ventricular function curve occurred at the levels of mitral regurgitation studied in Figure 4-5. If ventricular distensibility remained essentially unchanged, then the above observations can best be explained by postulating more complete systolic emptying when this degree of mitral regurgitation is present. This view is also supported by the data shown in Figure 4-6 in which, with filling

pressure held constant, the ventricle could eject substantially larger volumes through the regurgitant pathway than into the aorta. More complete systolic emptying is facilitated not only by the high-pressure gradient between ventricle and atrium but also by the continuation of regurgitation during proto-diastole and isometric relaxation as shown by Wiggers.⁶ It is clear, however, that the primary compensatory mechanism of more complete systolic emptying is gradually exhausted when mitral regurgitant stroke volumes increase. Under such circumstances, an increase in end-diastolic ventricular volume must occur. This is indicated not only by the slight depression of the ventricular function curve, which is always observed, but also by progressive depression of the ventricular function curve as mitral regurgitant volumes are further augmented.

It was not anticipated that the mitral regurgitant flows produced would be as high as those observed⁶ nor that they would be associated with such small elevations of left atrial pressure, since more striking elevations of this pressure are observed when mitral valve injury is experimentally induced^{6,7} and also in the naturally occurring disease state in man.⁸ Further, if left ventricular hypertrophy is associated with mitral regurgitation, in the absence of other myocardial disease, it might be anticipated that the volume of regurgitation the ventricle can endure would be greater than in the absence of such hypertrophy.

The observation that the effect of any given regurgitant volume on left atrial pressure is a function of the filling pressure prior to the induction of regurgitation may be helpful in the interpretation of hemodynamic findings in clinical mitral insufficiency. With the heart operating on any given left ventricular function curve,³ its specific position on that curve is of importance, as can be seen in Figure 4-7. That is, when regurgitant flows are induced with the preregurgitant filling pressure initially low, the consequent rise in left atrial pressure is substantially less than when the same range of regurgitant flows were induced with the ventricle higher on its

ventricular function curve, i.e., with a higher filling pressure. Further, the particular ventricular function curve on which the heart is operating will substantially influence the filling pressure at which the ventricle is doing its effective stroke work. That is to say, if the left ventricle is, because of disease, operating on a depressed function curve, the filling pressure and left atrial pressures will be higher at any given stroke work. Under such circumstances, the effect of any given mitral regurgitant volume will be to elevate left atrial pressure more than if the ventricular function curve were not depressed (Fig. 4-8).

The increase of left atrial pressure that occurs with any given regurgitant stroke volume will also be a function of that point on the pressure-volume curve of the left atrium and pulmonary vascular bed⁹ obtaining at that time. If, as recently suggested,¹⁰ changes in pulmonary vascular distensibility do occur, such changes may also modify the extent of the rise in left atrial pressure.

The observation of elevated left ventricular filling pressures in patients with pure mitral insufficiency,¹⁰ when viewed in the light of the above data, could be explained by the presence of (1) mitral regurgitant flows relative to cardiac output grossly in excess of those observed herein, (2) depressed ventricular function curves due to myocardial disease, or (3) possibly to changes in ventricular distensibility secondary to myocardial hypertrophy. Haynes *et al.*¹¹ using the Korner-Shillingford method¹² for the estimation of mitral regurgitant flow, observed regurgitant flows averaging 165 per cent of the forward cardiac output in patients with predominant mitral regurgitation in whom left ventricular end-diastolic pressure were not elevated.

Wiggers⁶ demonstrated striking elevations of the left atrial "V" wave when aortic pressure was increased in the presence of mitral regurgitation. The above data confirm the anticipation that mitral regurgitation is augmented under these circumstances, as was also found in the model studies and aortico-left atrial fistula preparation of Rodbard *et al.*^{13,14} Such augmentation of mitral regur-

gitant flow and the consequent striking elevation of the "V" wave when resistance to aortic ejection is increased has also been observed in patients with mitral regurgitation when aortic pressure was markedly elevated by nor-epinephrine infusion. This response has proved helpful as a diagnostic test for the presence of even mild degrees of mitral regurgitation which were not otherwise hemodynamically apparent.¹⁵

It has been demonstrated recently¹⁶ that the oxygen consumption of the heart is determined largely, if not solely, by the tension-time index (mean aortic systolic pressure times the duration of systole), and is independent of the stroke volume except insofar as the latter changes the duration of the tension state developed by the myocardium. Planimetric integration of the systolic pressure curve provided the tension-time index in three of the above experiments in which stepwise increases in mitral regurgitation were produced. No significant change in the tension-time index occurred. These data strongly suggest that the oxygen consumption of the left ventricle is not greatly increased by large volumes of mitral regurgitation and may help to explain the clinical observation that angina pectoris is rare even with severe mitral insufficiency.

Throughout the course of these experiments, acutely produced aortic regurgitation resulted in a hemodynamic pattern that consistently stood in sharp contrast to that produced by similar or even greater volumes of mitral regurgitation. The volumes of aortic regurgitant flow that were observed in these experiments do not appear to be unrealistic in terms of the clinical counterpart of this lesion. When in clinical studies on patients with severe aortic insufficiency dye was introduced into the aorta at or below the level of the diaphragm, it was observed to regurgitate retrograde to the innominate artery as indicated by its photometric detection in the ear promptly after the aortic injection.¹⁷ The studies of clinical aortic regurgitation by the method of Korner and Shillingford¹² would also appear to be consonant with the above views. With the development of compensatory

hypertrophy, it would be expected that as in mitral regurgitation larger aortic regurgitant volumes could be tolerated than when the lesion is acutely induced as in these experiments.

Unlike mitral regurgitation, aortic regurgitation was accompanied by severe depression of the effective left ventricular function curve (Fig. 4-10A,B). In contrast to the more complete systolic emptying of mitral regurgitation, the increased total stroke volume of aortic regurgitation is ejected into the aorta in which the pressure is higher than in the left atrium and progressively rises as ejection proceeds. It appears that the increased stroke volume of aortic regurgitation is accompanied by an increased end-diastolic volume.

The observed increase (Figs. 4-9 and 4-12) in calculated peripheral resistance, which accomplished the fall in effective cardiac output when aortic regurgitation was acutely induced, represents the activity of baroreceptor regulation in maintaining arterial pressure. Without such a constrictor response, diastolic coronary perfusion pressure would fall to levels even lower than those that are maintained. This constrictor response may, therefore, be construed as conferring a protective effect under these experimental conditions. Clinically, since cardiac output at rest is not generally decreased in patients with mild or moderate aortic regurgitation, there is no requirement for an increased peripheral vascular resistance. However, it might be anticipated that, as the severity of the regurgitant lesion progresses and effective cardiac output is depressed because of the ventricle's inability to further compensate, increased peripheral vascular resistance may play an important role in the maintenance of arterial and coronary perfusion pressures. With this in view, it is of interest that the administration of ganglionic blocking agents or spinal anesthesia may be hazardous¹⁸ in the patient with severe aortic regurgitation.

Ample confirmation was obtained in these experiments of the clinical observation by Wright *et al.*¹⁹ and Morrow²⁰ that left ventricular end-diastolic pressure may occasionally exceed left atrial pressures in the

presence of aortic regurgitation. The experiments shown above in Figure 4-12, in which mitral regurgitation was superimposed on aortic regurgitation, were designed to elucidate the possible physiological significance of this interesting phenomenon. First, it is clear that a competent mitral valve must close partially or completely well before the onset of systole, else the late diastolic reverse pressure gradient between ventricle and atrium could not develop. Secondly, in the presence of aortic regurgitation, compromising the competence of the mitral valve produced a further elevation of left atrial pressure. Simultaneously, the left ventricular end-diastolic pressure fell as shown in Figure 4-6. This decrease in left ventricular end-diastolic pressure (and presumably in fiber length) resulted in a further diminution of stroke work and cardiac output from already depressed levels. Under such circumstances the mitral valve may be thought of not only as a secondary resistance to regurgitant flow from the aorta, but also as a means of protecting the pulmonary vascular bed from elevated pressures, while permitting the left ventricle to achieve a high enough end-diastolic pressure and volume for the discharge of a more adequate stroke volume than would otherwise be the case. Such considerations apply, however, only if the ventricle is on the ascending limb of its ventricular function curve. These data also suggest that the inadvertent production or increase of mitral insufficiency in the course of mitral commissurotomy may have substantially profounder consequences in the presence of aortic regurgitation than in its absence. Uricchio and Likoff²¹ attributed the adverse effects of mitral commissurotomy in the presence of aortic stenosis and insufficiency largely to the aortic stenosis, which they felt was functionally intensified by mitral commissurotomy. The above data provide the basis for one possible alternative interpretation.

SUMMARY AND CONCLUSIONS

Experimental techniques were devised in the anesthetized open-chest dog by means of

which the regurgitation of blood from left ventricle to left atrium and from aorta to left ventricle could be metered and varied over wide ranges

Mitral regurgitant flows from zero to three times resting cardiac output were tolerated with only slight alterations of effective cardiac output, aortic, left atrial, and left ventricular pressures and total peripheral resistance. There was little depression of the effective left ventricular function curves with mitral regurgitant flows of approximately 2 liters per minute. Any given increase in mitral regurgitant flow required substantially smaller increments in ventricular filling pressure than did similar increases in effective cardiac output. These observations suggest that ventricular emptying is more complete in the presence of mitral insufficiency. Further observations revealed that, with any given mitral regurgitant orifice, regurgitant flow was a function of aortic pressure. In other experiments, left atrial pressures were initially elevated by producing high effective left ventricular work levels, and these work levels were held constant during progressively increased mitral regurgitation. Under such circumstances, mitral regurgitation did produce substantial increments in mean left atrial and left ventricular filling pressures. The extent of this rise, resulting from any given degree of mitral regurgitation, was a function of the mean left atrial and ventricular filling pressures prior to the induction of mitral regurgitation. These observations suggest the importance of the relationship between myocardial contractility and the hemodynamic effects of any given mitral regurgitant lesion.

Stepwise increases in aortic regurgitation were produced at a constant heart rate until regurgitant flows in excess of the dog's resting stroke volume were observed. A substantial decrease in effective stroke volume occurred. Total peripheral resistance rose markedly. Aortic pulse pressure always widened. The left ventricular function curve was markedly depressed in the presence of aortic regurgitation.

When, at a constant heart rate, aortic

regurgitation was induced in the presence of a competent mitral valve, left ventricular end-diastolic pressure (LVED) rose to much higher levels than left atrial pressure (L.A.). This difference in LVED and L.A. could be diminished or almost abolished by inducing concomitant mitral insufficiency. At such times, however, the fall in LVED was accompanied by a further diminution in effective stroke volume beyond that which had been produced by the aortic regurgitation alone.

Thus, in the presence of aortic regurgitation, a competent mitral valve acts in two ways to protect the circulation. First, it limits the elevation of left atrial and pulmonary capillary pressures. Secondly, it makes possible the high left ventricular end-diastolic pressure, as a result of which a more forceful ventricular contraction occurs, provided the ventricle is still on the ascending limb of its ventricular function curve.

ACKNOWLEDGMENTS

Acknowledgment is made to *Circulation Research* in which the following papers were published:

1. BRAUNWALD, E., WELCH, G. H., SARNOFF, S. J.: The hemodynamic effects of quantitatively varied experimental mitral regurgitation, *Circulation Res.* 5:539, 1957.
2. WELCH, G. H., BRAUNWALD, E., SARNOFF, S. J.: The hemodynamic effects of quantitatively varied experimental aortic regurgitation, *Circulation Res.* 5:546, 1957.

REFERENCES

1. SARNOFF, S. J., and BERGLUND, E.: Ventricular Function I. Starling's law of the heart studied by means of simultaneous right and left ventricular function curves in the dog, *Circulation* 9:706, 1954.
2. SARNOFF, S. J., DONOVAN, T. J., and CASE, R. B.: The surgical relief of aortic stenosis by means of apical-aortic valvular anastomosis, *Circulation* 11:564, 1955.
3. SARNOFF, S. J., and BERGLUND, E.: The Potter electroturbineometer. An instrument for recording total systemic blood flow in the dog, *Circulation Res.* 1:331, 1953.

- 4 WELCH, G. H., BRAUNWALD, E., MACRUZ, R., and SARNOFF, S. J.: The effect of mephentermine sulfate (Wyamine) on myocardial oxygen consumption, myocardial efficiency and peripheral vascular resistance, *Am J Med* 24:871, 1958.
- 5 BRAUNWALD, E., SARNOFF, S. J., and STAINSBY, W. N.: Determinants of duration and mean rate of ventricular ejection, *Circulation Res* 6:319, 1958.
- 6 WIGGERS, C. J., and FEIN, H.: The cardiodynamics of mitral insufficiency, *Heart* 9:141, 1921-1922.
- 7 HALLER, A. J., JR., and MORROW, A. G.: Experimental mitral insufficiency, *Surgery* 38:518, 1955.
- 8 MORROW, A. G., BRAUNWALD, E., HALLER, J. A., JR., and SHARP, E. H.: The left atrial pressure pulse in mitral valve disease: A correlation of pressures obtained by transbronchial puncture with the valvular lesion, *Circulation* 16:399, 1957.
- 9 SARNOFF, S. J., and BERGLUND, E.: Neurohemodynamics of pulmonary edema IV: Estimated changes in pulmonary blood volume accompanying systemic vasoconstriction and vasodilatation, *Am J Physiol* 170:568, 1952.
- 10 ROSS, J. R., JR., BRAUNWALD, E., and MORROW, A. G.: Clinical and hemodynamic studies in pure mitral insufficiency, *Am J Cardiol* 2:11, 1958.
- 11 HAYNES, F., NOVACK, P., SCHLANT, R., PHINNEY, A., and DEXTER, L.: Hemodynamics of mitral stenosis and regurgitation, *Fed Proc* 16:56, 1957.
- 12 KÖRNER, P. I., and SHILLINGFORD, J. P.: Quantitative estimation of valvular incompetence by dye-dilution curves, *Clin Sc* 14:553, 1955.
- 13 RODBARD, S., and WILLIAMS, F.: The dynamics of mitral insufficiency, *Am Heart J* 48:521, 1954.
- 14 RODBARD, S., KRAUSE, E., LOWENTHAL, M., and KATZ, L. N.: Acute dynamic effects of a shunt between a systemic artery and the left atrium, *Am J Physiol* 187:438, 1956.
- 15 BRAUNWALD, E., WELCH, G. H., and MORROW, A. G.: The effects of acutely increased systemic resistance on the left atrial pressure pulse: a method for the clinical detection of mitral insufficiency, *J Clin Invest* 37:35, 1958.
- 16 SARNOFF, S. J., BRAUNWALD, E., CASE, R. B., STAINSBY, W. N., WELCH, G. H., JR., and MACRUZ, R.: Oxygen consumption of the heart, *Fed Proc* 16:112, 1957.
- 17 BRAUNWALD, E., and MORROW, A. G.: The estimation of aortic regurgitant flow in man, *Circulation* 17:505, 1958.
- 18 SARNOFF, S. J., and CASE, R. B.: "Physiological Considerations Relative to the Hufnagel Operation with Special Reference to Post-operative Anemia," *Symposium on Cardiovascular Surgery, Henry Ford Hospital Philadelphia*, W. B. Saunders & Co., 1955, p. 304.
- 19 WRIGHT, J. L., TOSCANO-BARBOZA, E., BRANDENBURG, R. A.: Left ventricular aortic pressure pulses in aortic valvular disease, *Proc. Mayo Clin* 31:120, 1956.
- 20 MORROW, A. G.: Personal communication.
- 21 URICCHIO, J. F., and LIKOFF, W.: Effect of mitral commissurotomy on coexisting aortic valve lesions, *New England J Med* 256:199, 1957.

CHAPTER 5 CORONARY BLOOD FLOW AND CARDIAC ENERGETICS

Steven M. Horvath

Although many investigators have been actively engaged in studying the coronary circulation, the precise role and relative importance of the factors involved in the regulation of the volume of blood flowing through the myocardium are still incompletely understood. None of the methods currently available provides for detailed or even accurate information regarding the coronary system in normal or diseased states. The four major experimental approaches to evaluating the coronary circulation involve studies on isolated coronary vessels, on isolated strips of cardiac muscle, on isolated or exposed hearts (in vivo and in vitro), and on the heart in the intact state. The two latter procedures are handicapped by, in the first instance, the application of highly complex precision instrumentation to an isolated or exposed heart already placed under considerable stress by the necessity for surgical intervention and the other by the employment of indirect methodology that does not provide for sufficient information on the entire heart. The heart-

lung preparations or heart oxygenator systems provide for certain technical advantages but only acute processes and changes can be followed. Furthermore, one is forced to generalize from the results obtained, assuming that stimuli that act on isolated heart in vitro must have the same effect on the heart in situ, where the heart is exposed to these stimuli not for minutes or hours but for months or years. However, despite these difficulties it has been possible by a careful synthesis of the accumulated data on metabolic and hemodynamic factors to obtain an insight into the mechanisms concerned with the maintenance of adequate coronary blood flow to the myocardium under a variety of conditions.^{1, 2}

The anatomic and dynamic details of the coronary circulation have been fairly well delineated. The pattern of flow of coronary blood from aorta to right atrium follows the general pattern of distribution noted in any other vascular bed. The epicardial branches of the coronary arteries and veins anastomose

with each other and also with extracardiac arteries and veins. Arteriovenous shunts are numerous. In addition to these typical patterns, the arterioles, capillaries, and superficial veins connect directly with both ventricular cavities by deep drainage channels, the arterio-luminal, the arteriosinusoidal, and Thebesian vessels. These latter are apparently not utilized to any great extent despite the presence of favorable dynamic conditions. Approximately 80 per cent of the blood entering the left coronary artery drains into the coronary sinus, while the remainder enters the right atrium from superficial cardiac veins. These vessels also drain some 80 to 90 per cent of the right coronary inflow into the right atrium. The coronary sinus receives the remainder of the right coronary artery flow.

DISTRIBUTION PATTERNS

The mass of myocardium supplied by each of these vessels is not constant. Schlesinger⁴ has identified three major distribution patterns. They are roughly classified on the basis of the myocardial areas supplied predominantly by the major coronary arteries. Approximately 50 per cent of hearts have right coronary preponderance, i.e., the right coronary artery provides blood to a larger area of the heart than does the left coronary artery. Some 17 per cent of hearts have left coronary preponderance and the remainder of hearts studied have a balanced distribution. This relative distribution varies with the species of animal investigated. Therefore, studies on the coronary circulation in normal and diseased hearts must take into consideration these different distributions. This has not always been done and has led to some difficulties in interpretation of data presented by various workers. Another factor that has not received much attention is the relationship of the origin and initial length of the coronary arteries to the large vessels leaving the heart. This intimate relationship may result in a temporary or even permanent constriction or kinking of these small arteries by changes in volume or position of the aorta and/or pulmonary artery. The consequent

reduction in coronary blood flow may result in angina, electrocardiographic changes indicating coronary insufficiency, and even acute myocardial necrosis.^{4,5}

INTERCORONARY ARTERIAL CHANNELS

One of the most important considerations of the anatomic distribution of the coronary vascular bed has to do with the presence or lack of intercoronary arterial channels. It has been estimated that potentially adequate collateral anastomoses are present in some 9 per cent of all hearts. This prophylactic measure of setting up and even enlarging anastomoses between coronary arteries is initiated by some hearts without the stimulus of coronary occlusion or insufficiency. In certain disease entities these intercoronary collaterals develop far beyond the degree observed in normal hearts, and even approach those observed following coronary artery occlusion or narrowing. Anemia, cor pulmonale, cardiac hypertrophy, or valvular disease appear to provide potent stimuli to the formation of new anastomoses. Compensatory changes of this type occur following coronary occlusion. Following coronary artery occlusion, the ventricles fibrillate (irreversible) or, if the hearts survive this episode they remain hypodynamic with limited work tolerance and marked cardiac pain. It is assumed that such hearts have too little collateral development for the amount of infarcted tissue. The hearts that survive exhibit a great increase in intercoronary arterial vessels, in terms of number, diameter and length. This increased collateral flow may originate through pre-existing nonfunctional collaterals or newly formed channels. This development is relatively slow in contrast to the rapid opening up of anastomoses following occlusion of carotid or femoral arteries. A number of experimental procedures designed to increase the intercoronary arterial channels have been reported. All these methods tend to increase primarily the anastomotic efficiency rather than provide for backward perfusion. Among the relatively successful experimental approaches effecting this development, probably working because

of the inflammatory reaction they induce, the following appear to offer the most effective approaches—sham cardiac operations, ligation of and/or arterialization of the coronary sinus, application of extracardiac tissue to myocardium, production of anemia, and application of mechanical and chemical irritants to the myocardium. These interventions apparently provide for a significant degree of protection against infarctions produced subsequently.

These results do not necessarily imply that application of these techniques will provide positive benefit to human beings having occlusive coronary artery disease. Furthermore, the value and success of these surgical procedures in improving upon the natural maximal collateral development following an infarction remains to be determined. A major point that needs to be evaluated in these cases is whether or not the surgical intervention can be initiated sufficiently soon after an infarction or occlusion has occurred to accelerate or augment the natural protective increase in collateral development. It has also been suggested that these procedures may have prophylactic value in those individuals having a strong family history of coronary disease. Until techniques can be devised that will measure the degree of actual or potential intercoronary arterial connections present in any heart, this would seem to be a rather premature approach on the basis of existing knowledge. However, this is an area of investigation that deserves vigorous pursuit.

The coronary circulation exists simply to provide the myocardial cells with a supply of materials to maintain the level of the metabolic pool and to provide sufficient oxygen to ensure an energy supply adequate to meet all demands. There is no reason to anticipate that the metabolic organization, i.e., *energy production, of the heart differs in any large qualitative manner from other tissues*, although there may be differences in degree that adapt the heart to its particular task. Catheterization studies in both dog and man have proved that the heart can use a variety of foodstuffs for its fuel. There is a further suggestion that in pathological situations cer-

tain fuel materials are chosen preferentially. The heart of a diabetic patient, for example, appears to derive most of its energy from fat with a decreased uptake of carbohydrates.

OXYGEN SUPPLY AND CONSUMPTION

The major concern of the coronary circulation, therefore, seems to be that related to the adjustments which ensure adequate oxygen supplies. These can be provided for either through alterations in volume of blood flow and degrees of oxygen extraction or by adjustments of mechanical and biochemical properties of cardiac cells so that a given myocardial oxygen uptake results in greater external cardiac work. Normally cardiac tissues are extraordinarily efficient extractors of oxygen from the perfusing blood (Table 5-1).

TABLE 5-1 NORMAL VALUES FOR HUMAN HEARTS BASED ON CATHETERIZATION STUDIES

Heart rate, per minute	80
Mean arterial pressure, mm. Hg	95
Mean pulmonary arterial pressure, mm. Hg	15
Total peripheral resistance, dynes/cm. ² /sec.	1,200
Pulmonary vascular resistance dynes/cm. ² /sec	230
Oxygen uptake, ml/M ² /min	134
Cardiac index, liters/M ² /min	3.6
Left ventricle work, Kg. M/M ² /min	4.6
Right ventricle work Kg. M/M ² /min	0.9
Oxygen content coronary sinus blood, vol per cent	5.3
Arterial-coronary sinus O ₂ difference, vol per cent	12.1
Arterial-mixed venous O ₂ difference, vol per cent	4.5
Coronary blood flow, ml/100 gm. L.V./min	74
Coronary MRO ₂ , ml/100 gm. L.V./min	8.6
Coronary vascular resistance, units	1.3
Body respiratory quotient, units	0.78
Left ventricle respiratory quotient, units	0.88

the coronary arteriovenous oxygen differences being approximately 12 vols per cent or even more. This leaves only a very small additional quantity of oxygen that can be efficiently extracted. The extent to which this extra quantity of oxygen could contribute to the total oxygen supply under conditions of stress is not completely known. Under some conditions it may be an important factor in

TABLE 5-2 SOME REPRESENTATIVE CHANGES IN CORONARY FLOW AND OXYGEN EXTRACTION AS MEASURED IN MAN*

	Coronary blood flow	O Extraction
Systemic B P (elevated)	↑↑	= ↑
Acute pulmonic or aortic stenosis	↑	↑
Chronic cardiac failure	↓ = ‡	↑
Hyperthyroidism	↑	↓ = ‡
Cigarette smoking	↑	↓
Anemia	↑	↑
Congestive heart failure	↓	↑
Coarctation of aorta	↑	↑
Aortic insufficiency	↑	↑
Epinephrine	↑	↓
Mitral valvular disease	=	↑
Hypertension	=	=

* Catheterization of the coronary sinus and the use of the nitrous oxide technique for so determining blood flow

↑↑ indicates increase

‡ = indicates no change

↓↓ indicates decrease

successful adaptation (Table 5-2). There is need to know more about this extraction efficiency, especially in conditions of natural maximal stress such as exercise in both normal and pathological subjects. Knowledge of the oxygen content of coronary sinus blood or the coronary arteriovenous difference is, therefore, essential. Such information enables a differentiation to be made between dilatation consequent to increased metabolic requirements by the heart or dilatation arising from changes in activity states of smooth muscle cells in vessel walls or extravascular factors operating on the vessels. If coronary flow increases and the arteriovenous oxygen difference decreases, the heart obtains its blood flow at least cost. When the stress situation requires greater blood flow and greater extraction of oxygen, then the heart is closer to the critical verge of utilizing nearly all its reserve capacities.

The economy of oxygen consumption by the ventricle has not been easy to determine. The basic problem here, as in the case of other muscular tissues, is to determine the

resting oxygen utilization. This value is essential to any calculations of myocardial efficiency. Some recent studies on the oxygen consumption of asystolic ventricles suggest that this value is relatively high, approximately half of that utilized by the beating heart. If these are confirmed, then calculations for cardiac efficiency are going to be greatly altered. Mechanical efficiency of the heart represents the percentage ratio of the work performed by the heart to the energy cost (oxygen usage) expressed in energy units. The usual value given for efficiency of the left ventricle is 25 per cent. A decrease in efficiency can result from either a decline in cardiac work or an increase in oxygen usage. However, a decreased myocardial efficiency does not necessarily indicate the presence of myocardial failure. It does indicate that with time the effects of mechanical failure will become evident. In patients with myocardial failure the fall in mechanical efficiency does not result from the increase in energy cost of heart work alone. The decrease in work done by the heart also induces a decrease in mechanical efficiency.

Inherent in any consideration of oxygen uptake by myocardial tissue is the question of the capacity of these cells to acquire an "oxygen debt." It has always been assumed that the heart cannot have an "oxygen debt" but some new evidence implies that this is not an impossibility. If these data are sustained, then the basic processes in cardiac muscle will fall more in line with the current concepts regarding striated muscle.

Reduction in oxygen consumption by the cardiac tissue is not synonymous with myocardial anoxia. Anoxia is present only if mean capillary or tissue oxygen partial pressure is reduced. The severity of metabolic disturbances depends simply on the ratio of oxygen demand by heart muscle and available oxygen supply. If both decrease the effects of metabolic ischemia will be slight or even absent. Excellent examples of this situation are seen in hypothyroidism, hemorrhagic hypotension, and during hypothermic states.

In the previous consideration of oxygen

utilization by the myocardium it is obvious that extraction ratios and volume of flow are closely interrelated.* Since it is not possible to increase the extraction of oxygen appreciably, alterations in volume of coronary flow could be the most effective manner of providing adequate supplies of oxygen. The factors determining coronary blood flow are incompletely understood. In fact, there are many questions as to the role of the multitudinous determinants that have been postulated. In all probability all these factors affecting coronary blood flow are not direct determinants but rather act in a complex integrated fashion. Among the more than twenty determinants which one or more investigators have implicated as having a direct relation to the volume of coronary flow only a few can be considered. It is, *a priori*, apparent that the effective perfusion pressure head and the size or mean bore of the coronary beds must constitute the main factors in the coronary circulation.⁶ This does not mean that the absolute level of the aortic pressure has any direct relationship to the coronary flow. Alterations in the diameter of the coronary vessels are consequent to passive or active factors. The passive mechanisms can work from within through a change in intravascular pressures or from without consequent to the myocardial wall, the extravascular support. In this respect it is interesting to note that the pendulum has swung around completely. The observations by Scaramucci,⁷ 1689, that a reduced amount of blood flows through the coronary vessels during systole owing to their compression by the contracting cardiac muscle appears now to be fairly acceptable. This is also in essential agreement with the observed decrease in striated muscle blood flow during its contraction. The active factor in changing diameters of coronary vessels is related to the state of the intrinsic smooth muscles in these vessels. If they relax, the resistance is decreased and an increased flow results even

though the effective perfusion pressure remains unchanged. The activity of these muscles is modified by metabolic, hormonal, and neural factors. Although much has been written regarding these mechanisms, their precise mode and degree of action remains undetermined.

The application of the accumulated knowledge regarding the coronary circulation to the intact human and dog heart has not always been possible, owing to difficulties in transferring open chest and isolated heart experimental data to an intact organism. The demonstration that the nitrous oxide technique for determination of cerebral blood flow could be employed to measure blood flow in the intact heart was a potent stimulus to the investigators of cardiac physiology and pathology.^{8,9} Although admittedly not a measure of the total blood flow through the heart, it does provide a sampling of the flows entering the myocardium through both major coronary arteries. It also, perhaps unfortunately, determines primarily the flow through the left coronary and by inference the blood coursing through the left ventricular myocardium. This technique, requiring catheterization of the coronary sinus, has not only provided the cardiologist with many new ideas but has led to tentative confirmation of the applicability of much of the information obtained by other experimental approaches. It is not feasible to list all the data or even consider their value to an understanding of the coronary circulation, but a short list of some changes occurring in the human heart are to be found in Table 5-2.

In this brief review, many of the other factors that can be considered as determinants of the coronary blood flow have had to be regrettably ignored. The influences of the heart rate, of the cardiac output, of hemodynamic factors other than mechanical factors, of neural, of chemical shifts in the myocardium, and the perfusing blood and of the multitudinous hormonal agents are not necessarily minor. They are sufficiently complex so that no simple statement of their role can be given. Undoubtedly, the validity of a previous statement, *i.e.*, that all these factors

* This may have to be evaluated in respect to the possibility that under certain pathologic conditions there is an increase in the amount of perfused nonmuscular tissue.

work in some co-ordinated integrative fashion, is the most precise way of indicating their contribution. There is a bright future ahead in our understanding of this complex mechanism and the development of new and ingenious approaches to the functioning of the intact heart lends support to the belief that soon the intricacies of the heart, normal and pathologic, will be revealed.^{10,11,12,13,14}

BIBLIOGRAPHY

- 1 GREGG, D E, and SABISTON, D C, JR.. Current research and problems of the coronary circulation, *Circulation* 13 916, 1956
- 2 GREGG, D E. *Coronary Circulation in Health and Disease*. Philadelphia, Lea & Febiger, 1950
- 3 SCHLESINGER, M J: Relation of anatomic pattern to pathologic conditions of the coronary arteries, *Arch Path* 30 403, 1940
- 4 DONALD, D E, and ESSEX, H E. Canine septal coronary artery, anatomic and electrocardiographic study, *Am J Physiol* 176 143, 1954
- 5 CORDAY, E, GOLD, H, and KAPLAN, L. Coronary artery compression: an explanation for the cause of coronary insufficiency in pulmonary hypertension, *Trans Am Coll Cardiol* 7:93, 1957
- 6 KATZ, A M, KATZ, L N, and WILLIAMS, F L. Regulation of coronary flow, *Am J Physiol* 180 392, 1955
- 7 SCARAMUCCI, DIARIO-MARPEUSE, 1689. Quoted by HALLER, ALBERT. *Elements Physiologiae Corporis Humani*. Lausanne, 1778, Vol. 4, p 459
- 8 GOODALE, W T, LUBIN, M, ECKENHOFF, J E, HAFKENSCHIEL, J H, and BANFIELD, W C, JR. Coronary sinus catheterization for studying coronary blood flow and myocardial metabolism, *Am J Physiol* 152 340, 1948
- 9 BING, R J. The metabolism of the heart, *Acta cardiol* 10 1, 1955
- 10 BING, R J: Role of the coronary circulation in shock, *Ann New York Acad. Sc.* 55 367, 1952
- 11 LEIGHT, L, DEFazio, V., TALMERA, F. N. REGAN, T J, and HELLEMS, H K: Coronary blood flow, myocardial oxygen consumption, and myocardial metabolism in normal and hyperthyroid human subjects, *Circulation* 14:90, 1956.
- 12 BARGERON, L M, JR, EHMEKE, D, GONLUBOL, F, CASTELLANOS, A, SIEGEL, A, and BING, R J. Effect of cigarette smoking on coronary blood flow and myocardial metabolism, *Circulation* 15 251, 1957
- 13 BING, R J, HAMMOND, M M, HANDELSMAN, J C, POWERS, S R, SPENCER, F C, ECKENHOFF, J E, GODDALE, W T, HAFKENSCHIEL, J H, and KETY, S S. Measurement of coronary blood flow, oxygen consumption, and efficiency of left ventricle in man, *Am Heart J* 38 1, 1949
- 14 HORVATH, S M, FARRAND, A, BLATTEIS, C, and EVERINGHAM, A. Catheterization of the coronary arteries of intact dog, *Am Heart J* 54 138, 1957

CHAPTER 6 CONTROL OF CARDIAC OUTPUT IN MAN

James V. Warren

During recent years much information has been obtained regarding the factors influencing cardiac output in man. It is of interest to view this development in chronologic perspective. For a considerably longer period of time, observations have been accumulating regarding cardiac output in the experimental animal. From these have emerged such important generalizations as Starling's law of the heart. Against this background the recent observations on intact man have appeared. These have been made possible to a large degree by the use of the intravascular catheter and the development of at least two satisfactory methods for determining cardiac output (application of the direct Fick principle, utilizing oxygen data and the dye injection method). In some regards the information about man has confirmed the earlier findings, but in other aspects apparent conflicts have been found. At the present time, it appears that we are entering a phase of integration of these data in an attempt to develop a better over-all understanding of the mechanisms affecting cardiac output.

IMPORTANCE OF CARDIAC OUTPUT

Before discussing the factors influencing cardiac output, it might be well to consider the position of this function in the circulatory hierarchy. What on first thought appears to be a dominant feature of circulatory activity is, from a more considered point of view, a somewhat secondary factor in regulation of the circulation. It is, in effect, the servant of other physiologic functions. The primary reason for being of the circulation is the feeding of the various capillaries in the body with an active flow of blood. It is in the capillary that the real business of the circulation is carried on with various factors, either local or general, controlling the amount of flow. The adequate function of such a system, however, is dependent upon a stable perfusing pressure, which brings us to a second level of control, namely, that of the arterial blood pressure. This in turn is predominantly set by the interaction of the peripheral resistance and the cardiac output. One could summarize, then, by saying that the cardiac output is

adjusted to a given situation when (1) the flow of blood to the various parts of the body is adequate to meet physiologic demands, and (2) it is adequate to maintain the arterial pressure within the limits defined by the normal action of baroreceptors. Attention in this discussion will at times be focused on the second factor, but one must not forget that supplying of an adequate blood flow to the body is undoubtedly the primary function.

Enthusiastic analysis of the individual components of the circulation should not lead us from the basic fact that we are dealing with a circular system of tubes without beginning or end. Cardiac output, therefore, in the normal individual, is quantitatively the same as pulmonary blood flow or systemic blood flow. There may, however, be transient inequalities in flow. For instance, as one goes from the horizontal to the vertical posture, the amount of blood in the pulmonary circuit decreases. This would require that, transiently, left heart output be greater than right. In the analysis of various experimental situations, one must be careful to determine whether he is dealing with a temporary adjustment circumstance or a more stable state of longer duration.

In the present discussion, rate and stroke volume will not always be separated for consideration. In many situations where increased circulatory demands are to be met, rate and stroke volume will increase hand in hand. In other situations where stroke volume may increase, secondary reflex alterations in rate may be in the opposite direction, occurring as part of the general hemostatic control of the circulation.

Probably the most striking feature of the observations on cardiac output in the intact man is that it is a highly variable function. Under relatively basal conditions and correcting the output for variations in individual body size, we may derive certain averages, but they have a fairly wide range of variation.^{1,2} One may enumerate many situations where the average level of cardiac output appears to deviate from these normal values. Let us select several illustrative examples from such a list and study them with respect

to physiologic principles known to affect the cardiac output in the experimental animal.

FLUID ADMINISTRATION AFTER DEPLETION

The circulatory state, *shock*, produced by depletion of blood volume, offers an interesting starting point. Here the experimental evidence indicates, in addition to a low blood volume, a low arterial pressure, a low pressure in the right atrium, and a low cardiac output. There is general agreement that replacement of the lost blood volume by almost any agent such as whole blood, plasma, or albumin solutions will raise the cardiac output concomitantly with an increase in right atrial pressure and the increase in total blood volume. The results from various laboratories are pleasantly uniform in this regard.^{3,4}

If we carry our infusion of intravenous fluid beyond the replacement of inadequate blood volume and rapidly infuse the normal subject, we find ourselves in a situation where the reported observations do not agree. In some laboratories increases in cardiac output in a relatively linear fashion were found as the blood volume and right atrial pressure were increased by the infused fluid.⁴ In others, although atrial pressure rose, the results were erratic.⁵ On personal discussion with authors involved in some of these papers, I have come to the belief that an infusion of fluid into a normal subject will produce an increase in cardiac output in perhaps 50 per cent of subjects, and the output will decline as the effects of the infusion wear off. More recent studies on elevation of right heart pressure by so-called G suit (antigravity) compression failed to elevate cardiac output.⁶ It is my impression that a direct and adequate correlation with changes in atrial pressure or blood volume cannot be derived. Studies on right ventricular end diastolic pressure give similar results, unfortunately measurement of left atrial pressure has not, to my knowledge, been studied in such experiments. It may be well to point out here related observations on other effects of fluid administration. The pressure recorded in the superior vena cava, right atrium, right ven-

tricle, pulmonary arteries, and so-called pulmonary capillary pressure all rise in nearly parallel fashion.^{7,8} The evidence indicates that within considerable limits the gradient of pressure from one chamber to another is not altered. The net distending pressure of the ventricle is changed, however. Similar alterations in pressure occur upon removing blood, either actually or by pooling it peripherally with tourniquets. The term "filling pressure" is subject to misinterpretation in this context. In such situations end diastolic pressure may be elevated, but filling gradient may not be altered. Such considerations must not be forgotten in any explanation for the observed cardiac output responses. Let us turn to other situations before we attempt to incorporate these findings in a working hypothesis.

There have been several observations on the effect of the motionless upright posture on cardiac output and, in general, there is agreement that a reduction in cardiac output of approximately 20 per cent occurs.^{1,9} Because of technical problems, measurement of right atrial pressure at this time is fraught with some difficulty, but recent measurements with the use of a miniature manometer on the catheter tip indicate that the pressure in the right atrium is decreased.¹⁰ Observations in our laboratory have indicated that the application of an antigravity (G) suit will return the cardiac output to a value similar to that in recumbency.⁹

EFFECTS OF DRUGS

Observations have been reported from several sources on the effect of an increased heart rate induced by atropine.^{9,11} Relatively little change in stroke volume occurs. The cardiac output is increased, therefore, if observations are made soon after the drug is administered. Of recent interest in our laboratory are observations on the atropine effect when a subject is in the upright position. Here, despite even greater increases in heart rate, the cardiac output is not increased. Stroke volume falls considerably. The application of an antigravity suit at this time re-

stores, in part, atropine responsiveness in terms of cardiac output.

There are observations on the effects of epinephrine and other related drugs, such as Isuprel (Isoproterenol hydrochloride or sulfate), which demonstrate that the cardiac output may be increased under the influence of these compounds.^{12,13} The increase noted here is usually the result of a combined effect of increased stroke volume with an increase in heart rate. Postural studies similar to those carried out with atropine, but with Isuprel, show only slight lessened response in the upright position when compared with the results in the horizontal position.¹¹

HYPEREMIA

Some years ago in our laboratory observations were made on the effect of a large area of reactive hyperemia.¹⁴ This was produced by placing blood pressure cuffs high on the thigh of normal subjects, inflating them above arterial pressure, and studying the reactions of cardiac output during the few minutes following a 20-minute period of ischemia. Intensive reactive hyperemia develops in the leg; heart rate, stroke volume, and cardiac output increase precipitously. Right atrial pressure is either unchanged or falls slightly. The reaction is similar to that observed in patients with large arteriovenous fistulas which can be opened and closed by manual pressure.^{15,16} It is striking how rapidly the changes in heart rate and stroke volume come about.

EFFECTS OF EXERCISE AND OF MALFUNCTION

The cardiac output rises with exercise. It would appear that this represents a summation of many of the effects already discussed. The exercising muscle is, in certain regards, an arteriovenous fistula. Humoral and nervous factors are most certainly added. The relative contribution by increased stroke volume and by changes in heart rate is variable and may, in part, be a reflection of training.

We have considered essentially normal variations in cardiac output, but it may be of in-

erest briefly to consider the situation of certain forms of circulatory malfunction. We have already considered a form of shock. In congestive heart failure, observations have shown that the cardiac output may be low, or in other situations it may be relatively high.¹⁷ It would appear most logical to state that the cardiac output is inadequate, it does not meet the demands of the body. If this situation is acute and severe, such as after a massive myocardial infarction, the shock picture rather than the picture of congestive heart failure occurs. If the interference is not as marked and prolonged over a long period of time, the secondary phenomena of water retention, etc., occur and the clinical picture of congestive heart failure results. Another interesting point of circulatory malfunction is that of so-called vasodepressor syncope. The cardinal feature of this reaction is an acute decline in arterial blood pressure, associated with inadequate perfusion of the brain and sudden loss of consciousness.¹⁸ Observations on the circulation have shown that the fall in blood pressure is due, primarily, to a sudden decrease in peripheral resistance. The normal response to this does not occur and there is a failure of the cardiac output to compensate. Observations on the cardiac output have shown a value slightly lower than normal, but not grossly so. This would appear, therefore, to be an episode of acute disorganization of the circulation, with loss of the normal integrating mechanisms.

Is it possible to fit all these observations together into a working schema? They involve a wide variety of situations, both normal and pathologic. Although one cannot be entirely sure, the evidence would indicate that the observations on cardiac output that we have discussed do fit with the general premise that the cardiac output is the servant rather than the director of circulatory activities. Truly, restrictions in the heart's activities may limit blood flow, and mechanisms in the strictest sense other than tissue needs may alter circulatory activity, but the latter would appear to be the keystone of our design of operation. There are actually inadequate facts upon which to build a complete explanation of the

mechanics of cardiac output. We must, therefore, deal with partial views and make an attempt, with our incomplete information, to fit these data into the concepts derived from animal experimentation.

During recent years in our laboratory, we have become impressed by the role of the so-called central blood volume as one of the determinants of cardiac activity.¹⁹ It would appear that with the normal individual in the recumbent position, the central blood volume or, in other words, the volume of blood available to the heart for immediate usage, is adequate. Ventricular filling occurs rapidly and tachycardia is well tolerated. Adding more to the central blood volume at this time appears to have relatively little effect, as can be determined from transfusion experiments and observations on the effect of an antigravity suit. On the other hand, depleting the central blood volume as in blood loss and in postural changes will hamper circulatory activity. The pooling of blood with tourniquets or the motionless upright position causes a small decline in cardiac output. Furthermore, it has been pointed out that in the upright position ventricular filling is less rapid and the ability to withstand tachycardia is not good. These considerations lead us to the belief that an adequate central blood volume is an important limiting factor of cardiac function but is far from the only one.

Humoral agents such as epinephrine and the effects of nervous impulses can readily be demonstrated to alter cardiac output. The exact mechanism of these changes is not entirely clear, but the increase in cardiac output following such substances as epinephrine can be accomplished even in the face of diminished central blood volume and associated diminutions in atrial pressure. They appear to act directly upon the myocardium, augmenting stroke volume. Physical factors such as diminished peripheral resistance would also appear to be important in certain regards as controlling factors in stroke volume. Here the difference would be that of better ventricular emptying rather than related primarily to inflow.

If we cannot decipher this problem on the

basis of the results of human subjects, let us look at it in light of the background of animal experimentation that we have. Are these variants directly or indirectly all related to alterations in response, as predicted by the work of Starling and his associates? Consideration of this point leads us to observe, first, that the picture is complicated, and that it is difficult to discern an underlying mechanism. Overlying factors of a physical, humoral, and nervous sort bring about changes in output that complicate the picture. It is much as in our consideration of the control of respiration. It is generally accepted that there is a single underlying mechanism involved in respiratory control. Yet many, and probably most, minute-to-minute variations in respiration are dependent upon other factors. Although a basic controlling mechanism may be present, it is not necessarily the most frequently involved in varying the activity of a system. One might postulate, therefore, that the mechanism outlined by Starling underlies all the variables in stroke volume that we have observed. This may be true. The evidence on human subjects would fit this thesis, but does not prove it. The evidence at hand does not allow one to take a dogmatic position. It appears that there may be a certain set, perhaps by homeostatic mechanisms, of the level of ventricular activity at a given time, beyond which filling does not occur except in unusual situations, more rigid than one might expect from the basic Starling concept. This involves consideration of the mechanics of ventricular filling, and the discussions that are going on currently in literature regarding whether filling is merely a passive phenomenon from extracardiac pressure sources or whether it is related to certain recoil phenomena within the heart itself.²⁰

We have attempted to formulate a working hypothesis of the control of cardiac output in intact man. Although there are many observations available for the investigator to consider, inadequate data are at hand for a definitive answer. Clearly any analysis of this problem today may be disrupted tomorrow by the addition of a new and, as yet, undiscovered factor.

BIBLIOGRAPHY

1. STEAD, E. A., JR., WARREN, J. V., MERRILL, A. J., and BRANNON, E. S.: The cardiac output in male subjects as measured by the technique of right atrial catheterization: normal values with observations on the effect of anxiety and tilting, *J. Clin. Invest.* 24:326, 1945.
2. DOYLE, J. T., WILSON, J. S., LEPINE, C., and WARREN, J. V.: An evaluation of the measurement of the cardiac output and of the so-called pulmonary blood volume by the dye dilution method, *J. Lab. & Clin. Med.* 41:29, 1953.
3. BRANNON, E. S., STEAD, E. A., JR., WARREN, J. V., and MERRILL, A. J.: Hemodynamics of acute hemorrhage in man, *Am. Heart J.* 31:407, 1946.
4. McMIGHAEL, J., and SHARPEY-SCHAFER, E. P.: Cardiac output in man by direct Fick method; effects of posture, venous pressure change, atropine, and adrenaline, *Brit. Heart J.* 6:33, 1944.
5. WARREN, J. V., BRANNON, E. S., WEENS, H. S., and STEAD, E. A., JR.: Effect of increasing the blood volume and right atrial pressure on the circulation of normal subjects by intravenous infusions, *Am. J. Med.* 4:193, 1948.
6. EICH, R. H., STAIB, I., and WERTHEIMER, M.: The acute effects of simultaneously increasing central venous pressure and peripheral resistance on cardiac output, *Clin. Res. Proc.* 5:169, 1957.
7. DOYLE, J. T., WILSON, J. S., ESTES, E. H., JR., and WARREN, J. V.: The effect of intravenous infusions of physiologic saline solution on the pulmonary arterial and pulmonary capillary pressure in man, *J. Clin. Invest.* 30:345, 1951. *Invest.* 30:345, 1951.
8. HENRI, J. P., GAUER, O. H., SIEKER, H. O., and WENDT, W. E.: Pressure-volume relationship in the low pressure side of the cardiovascular system, *Am. J. Physiol.* 171:735, 1952.
9. WEISSLER, A. M., LEONARD, J. J., and WARREN, J. V.: Observations on the central venous reservoir as a determinant of cardiac output: effects of posture and atropine (To be published.)
10. SIEKER, H. O., and GAUER, O. H.: Study of postural effects on pressure relationships in the venous circulation, *Clin. Res. Proc.* 5:102, 1957.

11. McMICHAEL, J, and SHARPEY-SCHAFER, E. P.: Cardiac output in man by direct Fick method, effects of posture, venous pressure change, atropine and adrenalin, *Brit Heart J* 6 33, 1944.
12. WARREN, J V.. *Unpublished observations*
13. WEISSLER, A. M, WARREN, J V., and LEONARD, J J Observations on the determinants of cardiac output. effects of posture and isoproterenol (To be published)
14. STEAD, E A, JR, and WARREN, J V.: Cardiac output in man, an analysis of the mechanisms varying the cardiac output based on recent clinical analysis, *Arch Int Med* 80. 237, 1947
15. NICKERSON, J L, ELKIN, D C, and WARREN, J. V. Effect of temporary occlusion of arteriovenous fistulas on heart, stroke volume, and cardiac output, *J Clin Invest.* 30 215, 1951
16. EPSTEIN, F H, SHADLE, O W, FERGUSON, T B, and McDOWELL, M E • Cardiac output and intracardiac pressures in patients with arteriovenous fistulas, *J. Clin Invest* 32 543, 1953
17. STEAD, E A, JR, WARREN, J V, and BRANNON, E S. Cardiac output in congestive heart failure, analysis of reasons for lack of close correlation between symptoms of heart failure and resting cardiac output, *Am Heart J* 35 529, 1948
18. WEISSLER, A M, WARREN, J V, ESTES, E H, JR, McINTOSH, H. D, and LEONARD, J J Vasodepressor syncope: factors influencing cardiac output, *Circulation* 15 875, 1957.
19. SJOSTRAND, T Volume and distribution of blood and their significance in regulating circulation, *Physiol Rev.* 33 202, 1953
20. BRECHER, G A *Venous Return*. New York, Grune & Stratton, 1956

CHAPTER 7 REGULATION OF THE HEART RATE

D. B. Dill

Introduction

In healthy man the resting heart rate may vary from 40 to 80. In exercise its maximum sometimes reaches 210, commonly reaches 200. It is my purpose to discuss the factors that influence the heart rate. Most of the observations were made in the Fatigue Laboratories, Harvard University, during the period 1927-1940. The subjects generally were either men accustomed to laboratory procedures or dogs selected because of the willingness and pleasure with which they participated in our experiments. Supporting data, essential for generalization, have been derived from animal experiments reported in the literature.

Factors to be discussed include (1) metabolic rate, (2) nutritional state, (3) posture, (4) environmental temperature, (5) body temperature, (6) age, (7) body size, and (8) emotional state.

Metabolic Rate

When other conditions are controlled, the heart rate increases in an almost linear fashion with metabolic rate. This relation begins

to break down at high metabolic rates. After the heart rate has reached its ceiling there may be, for a short time, a further increase in metabolic rate by anaerobic utilization of stored energy reserves. The position and slope of the curve relating heart rate to metabolic rate varies widely from man to man. The trained athlete often has a lower resting rate than the nonathlete; he always has a smaller increment in heart rate for a given increment in metabolic rate.

The performances of four individuals are compared in Figures 7-1 and 7-2. All the subjects of this study were known to be in good health. One subject, DeMar, the Marathon runner, had maintained a state of physical training continuously for approximately 20 years. He ran ten miles or more daily, with few exceptions, throughout the year. The subject C.V.C. had never had extended experience in any form of physical exercise. Both D.B.D. and A.V.B. had led sedentary lives but have been accustomed at times to periods of hard physical work. The former was a runner while in college. The study of

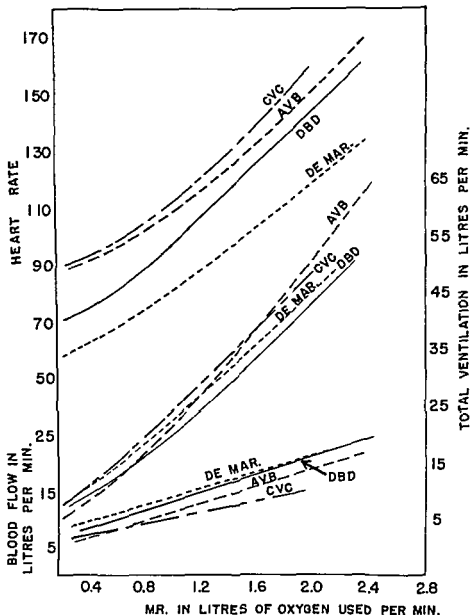


FIG 7-1 Comparative changes in four subjects of blood flow, total ventilation, and heart rate as functions of metabolic rate (Courtesy *Journal of Physiology*)

DeMar covered a period of two weeks, the experiments being done before breakfast. The data in the case of CVC were collected in a period of about six weeks, those on DBD and AVB in six months, the experiments generally being done 1 to 3 hours after breakfast.¹

In Figure 7-1 the heart rate in exercise depicts the physical state of the four subjects

When the oxygen consumption was 2.0 liters/min the most fit subject, DeMar, had a heart rate of 118 per minute while that of the least fit, CVC, was 160 beats per minute. The slow heart rate of the athlete appears to be directly related to the magnitude of the systolic output, or stroke volume, of his heart. Inspection of Figure 7-2 shows that the stroke volume at rest of the athlete

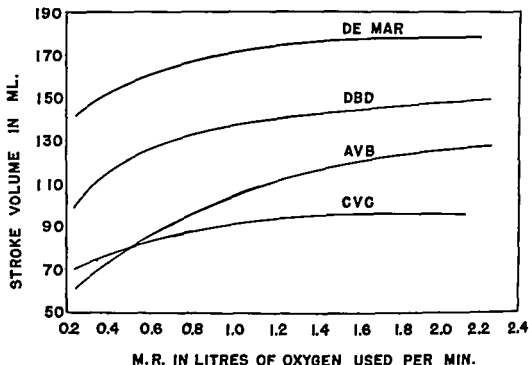


FIG. 7-2 Stroke volume in four subjects as a function of metabolic rate (Courtesy *Journal of Physiology*)

may be double that of the nonathlete subject. During work the stroke volume curve in the case of DeMar reaches an asymptotic level of 177 ml at a metabolic rate (M.R.) of 2.0 liters of oxygen used per minute. In CVC the resting stroke volume is about 70 ml and apparently reaches a maximum of 95 ml at M.R. 1.7. The two intermediate curves of DBD and A.V.B. probably more closely approximate the cardiac response of the average man of nonathletic pursuits.

The extent to which observations departed from the smoothed curves is shown for one subject in Figure 7-3. In a few instances the heart rate was 10 beats or more per minute greater or less than the smoothed values. It was partly this variability in response that led us to evaluate other factors that influence the heart rate.

Nutritional State

In man, well-controlled experiments take into account nutritional state. The resting heart rate is higher during digestion than in the postabsorptive state. The reaction to a

given bout of exercise depends to some extent on whether the subject has eaten recently. In long-continued work the heart rate may be affected by the lack of easily mobilized fuels; this has been demonstrated in the dog. Our dog, Joe, loved to run on the treadmill and would continue until exhausted. In one experiment, starting in the fasting state, he ran up a grade of 17.6 per cent at a rate of 155 m/min. (5.8 miles/hr.) for 4.75 hours.² His blood sugar declined from 80 to 50 mg per cent and his heart rate from 235 to 220 beats per minute. He was then stopped for five minutes and given 20 gm of sugar candy. He then began running again, during the next half hour his blood sugar increased to 100 mg per cent and his heart rate to 235 beats per minute. Further details are given in Figure 7-4.

Keys and associates³ have reviewed the literature on heart rate in man during starvation and rehabilitation. Tachycardia is a common feature of starvation. In the 32 subjects studied by Keys and his co-workers, the mean heart rate was 35.3 beats per

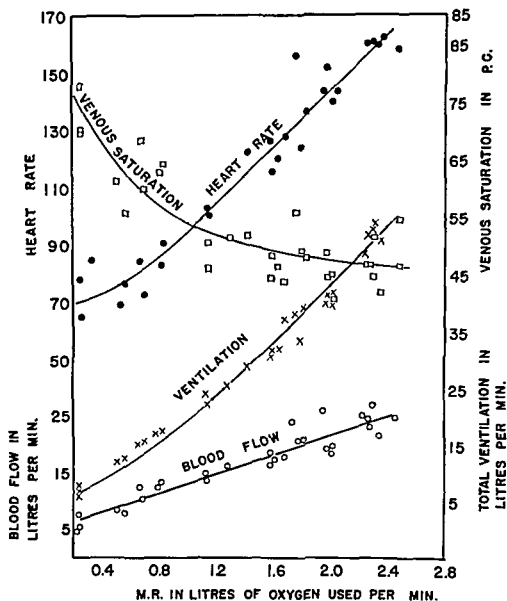


FIG 7-3 Blood flow, total ventilation, and heart rate of subject DBD as functions of metabolic rate (Courtesy *Journal of Physiology*)

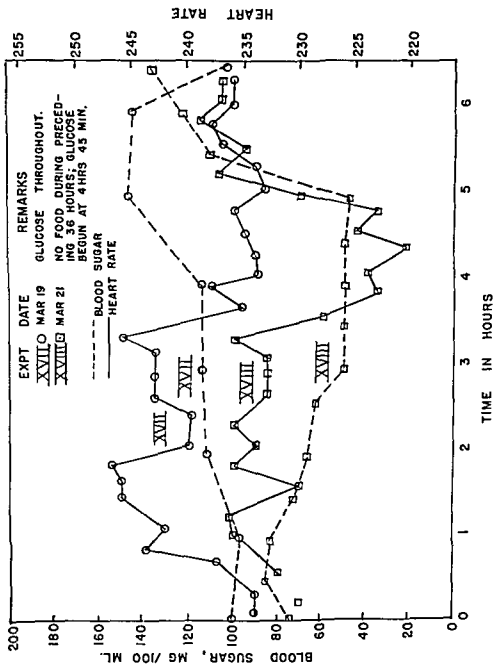


FIG. 7-4. Heart rate and blood sugar as related to exercise and intake of sugar. (Courtesy *Journal of Physiology*)

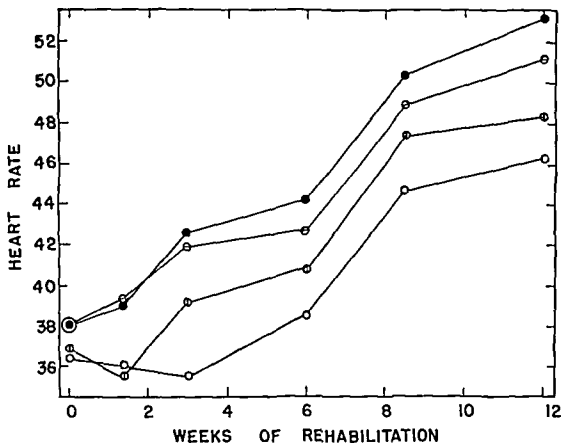


FIG 7-5 Mean basal heart rates of four caloric groups during the first 12 weeks of rehabilitation (Keys, A, *et al*, *The Biology of Human Starvation*, Copyrighted, 1950, University of Minnesota)

minute after three months on a low caloric diet. In two men the rate was 28 beats per minute. There was a slight increase during the next ten weeks on their inadequate diet. Recovery during rehabilitation did not begin until after the second week of rehabilitation, as is seen in Figure 7-5.

Posture

The heart rate during rest is related to posture. The heart beats more rapidly in the erect position than in the reclining position. The difference in rate varies from one individual to another and, generally, is smallest in physically fit individuals. This fact has been used, on occasion, in assessment of physical fitness. Table 7-1 contains observations on the heart rates and on venous oxygen saturation in the femoral vein in subjects standing and reclining.*

TABLE 7-1 EFFECT OF POSTURE ON HEART RATE AND OXYGEN SATURATION OF BLOOD IN THE FEMORAL VEIN

Subject	Venous oxygen % HbO ₂		Heart rate per min	
	Reclining	Standing	Reclining	Standing
Doorakian	90	65	78	75
Dill	76	43	56	60
Anderson	84	55	72	80
Jordan	77	31	62	75
Koerper	75	38	72	76
Bowen	65	35	79	88
Smith*	66	32	72	132
Bock	57	32	66	70

* Recuperating from gastric ulcer

Environmental Temperature

Environmental temperature is a major factor in determining the heart rate, especially

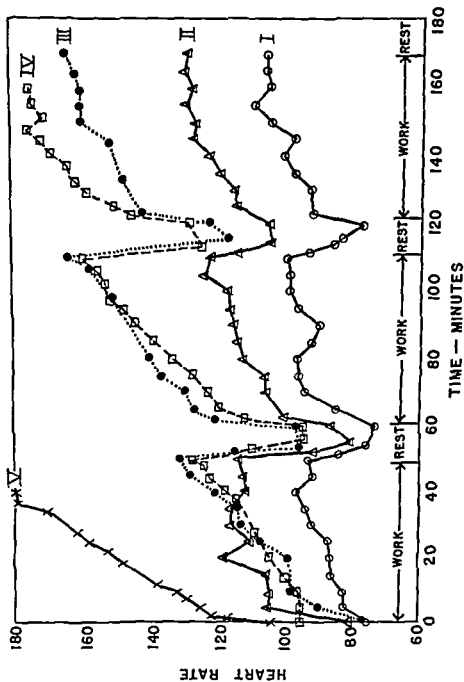


FIG 7-6 The effect of restricted evaporation of sweat on the heart rate. The metabolic rate (MR) in Experiments I-IV is about three times basal, in V, six times basal. Conditions were as follows:

- I Heavy woolen clothing worn
- II Face mask and rubber gloves in addition to woolen clothing.
- III. Olskin suit worn over woolen clothing
- IV Face mask, rubber gloves, and olskin worn in addition to woolen clothing.
- V. Similar to IV except for higher grade of work (Courtesy *Physiological Review*)

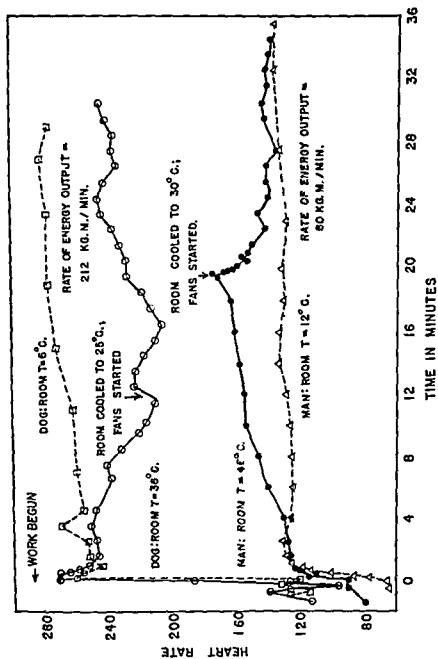


FIG 7-7 The effects of external temperature on the heart rates of man and dog during work. (Courtesy *Journal of Physiology*)

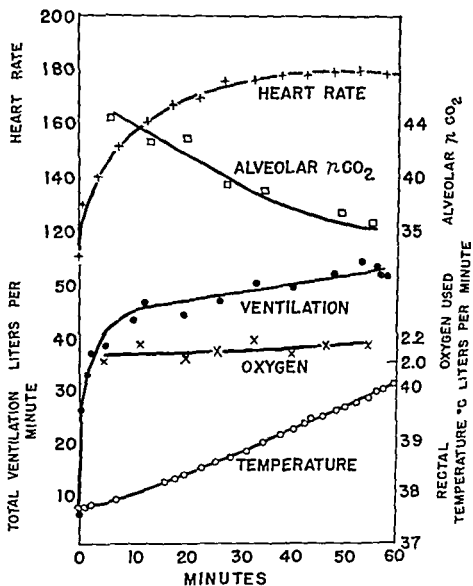


FIG. 7-8 The performance of a trained athlete working in high external temperature. (Courtesy *Arbeitsphysiologie*.)

ally in exercise of long duration. It is commonly stated that in moderate exercise a steady state is reached in two or three minutes. This certainly is not true when heat dissipation is difficult. In one series of experiments illustrated in Figure 7-6, a subject carrying a 40-pound pack walked on a treadmill at a room temperature of $25^{\circ}C$., but with combinations of clothing that interfered in varying degrees with heat dissipation.⁵ He walked for three consecutive 50-minute periods with intervening 10-minute rest periods

Perhaps the most striking feature of this series of experiments was the cumulative effect on the heart rate. Despite the constancy of oxygen consumption, the relative constancy of body temperature, and uniformity of experimental conditions other than the clothing, the heart rate increased in all experiments. The states of most variables concerned were as notable for their constancy as was that of the pulse for its lack of constancy. Trends in heart rate, which might have been considered of minor significance

had the experiments been terminated in 20 minutes, became a major phenomenon by the end of three hours.

One of the lessons to be learned from comparative physiological studies is caution in generalization. This is illustrated by the results shown in Figure 7-7.² In this series of experiments, it was found that man and dog respond quite differently to work in a hot environment, although their responses in the cold room were alike. As the man approached exhaustion at a high external temperature his heart rate increased, exhaustion coincided with the attainment of his maximum heart rate. At this point, fans were turned on the man and the room was cooled suddenly. Work was continued at the same rate, the heart rate decreased 30 to 40 beats per minute, and within 3 minutes the subject had passed from a state of exhaustion to one of comfort. The dog, on the other hand, showed a decrease in rate with the onset of exhaustion, and after the room was cooled, recovery, which was slower than in man, was accompanied by an increase in heart rate. Similar observations have been made on other occasions, evidently the con-

trol of the heart rate under these conditions is different in the two species.

In a series of experiments published in 1931,³ each of four subjects carried on the same grade of work at two widely separated temperatures, -12°C . and 34°C . The humidity was about 50 per cent in each case. The grade of work selected for each subject brought on exhaustion within 37 to 60 minutes in the hot room and was carried on easily in the cold room. Observations were made of rectal temperature, heart rate, alveolar pCO_2 , total ventilation, and oxygen consumption. The results on the most fit subject, an oarsman aged 43 years, are shown in Figure 7-8. His heart rate reached a plateau of 180 beats per minute after 40 minutes. His rectal temperature increased steadily, it had reached 40.1°C . when we terminated the experiment at 60 minutes.

Our studies led us to believe that experienced subjects in good health and under controlled conditions would show a predictable response in heart rate.⁴ Figure 7-9 and 7-10, prepared by L. J. Henderson, are two

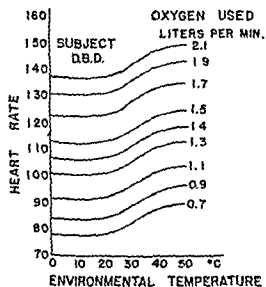


FIG. 7-10 The heart rate of FC as related to metabolic rate and external temperature (Courtesy American Heart Journal)

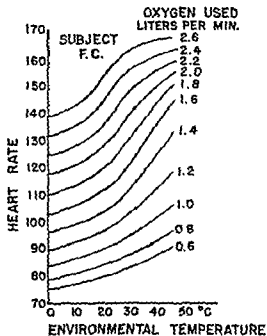


FIG. 7-10 The heart rate of FC as related to metabolic rate and external temperature (Courtesy American Heart Journal)

families of curves representing my heart rate and that of Frank Consolazio, my assistant. The external temperature was kept constant in a given experiment, and the heart rate was recorded after ten minutes of graded work on the bicycle ergometer. The temperature varied from 0° C. to 50° C., and the oxygen requirement from 0.6 to 2.6 liters per minute. The humidity at the higher temperatures was kept at about 50 per cent, and the air movement was minimal. The relations shown by these families of curves were extraordinarily reliable. Subsequent experiments proved that the probable error, within the range shown, was not greater than 2 or 3 beats per minute.

Body Temperature

Since the pioneering study by Smith and Fay,⁸ a great deal of attention has been given to the heart rate as related to body temperature. In hibernators the heart rate drops to one or two beats per minute after the body temperature has stabilized at 1° C to 3° C. Man cannot be cooled, safely, to temperatures below about 25° C. During the cooling process, unless anesthetics and other

medication are used, there will be shivering and elevation of the heart rate. Once a temperature of about 25° C. is attained, the heart rate drops, metabolism may be reduced to one-half basal and operations on the heart can be carried out with several minutes interruption of blood flow through it without damage.

In exercise, the effects of high external temperature on heart rate are so dominant that a direct relation between body temperature and heart rate is difficult to detect. For example, Figures 7-11, 7-12, and 7-13 illustrate paired experiments conducted at high and low temperatures. Figure 7-13 shows that for one man working at a fixed rate, there may be a wide difference in heart rates for the same rectal temperature. For example, in the case of this subject, with an oxygen consumption of 1.7 liters per minute, the rectal temperature attained 38.2° C after work for 40 minutes in the cold room and after work for 17 minutes in the hot room. The heart rates at these times of equal metabolic rates and equal body temperatures were 135 and 168 beats per minute respectively.

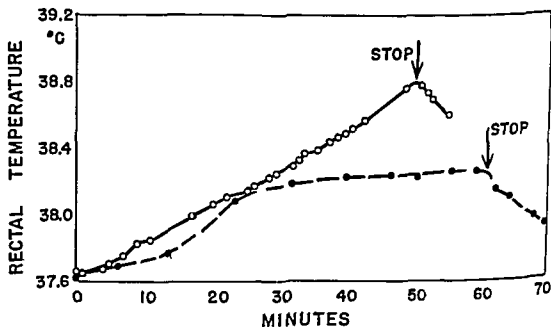
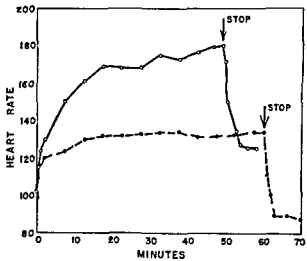


FIG. 7-11 The rectal temperature during work in high and low temperatures (Courtesy *Arbeitsphysiologie*)

FIG 7-12. The heart rate during work in high and low environmental temperatures (Courtesy *Arbeitsphysiologie*.)



Age
In the resting state, the heart rate of boys is higher than in man, and higher still, in exercise Robinson⁹ has studied the performance of a large number of subjects doing a fixed task on the treadmill, i.e., walking at 5.6 km per hour up a grade of 8.6 per cent. This can be accomplished by boys of 6 years or men of 70 years. The oxygen consumption is about seven times the basal rate. The heart rate after fifteen minutes of such work is much higher in boys than in men of middle age. Such work produces a mean heart

rate of 170 beats per minute in 6-year-old boys, as contrasted with 134 beats per minute in men 42 years of age (Table 7-2).

TABLE 7-2 HEART RATES DURING GRADE WALKING AS RELATED TO AGE
(The speed was 5.6 km per hour, and the grade, 8.6 per cent. This raises the oxygen consumption to about seven times basal.)

Age (years)	Heart rate (per min)
6	170
10	164
14	160
18	150
22	146
26	143
30	140
34	137
38	134
42	134

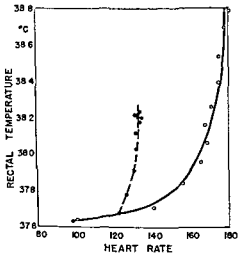


FIG 7-13 The relation between heart rate and rectal temperature during work in high and low environmental temperatures (Courtesy *Arbeitsphysiologie*.)

Young dogs have high heart rates, these appear to reflect great sympathetic stimulation. It has been shown by Brouha and his associates that, after the dog has been sympathectomized, his performance during moderate exercise may remain unaffected, insofar as fatigue, accumulation of lactic acid, and mobilization of energy reserves are concerned, and yet a given task will be accomplished with a much slower heart rate, and, therefore, with a much larger oxygen pulse. It appears that, before the operation, the heart rate is unnecessarily high. These

facts suggest that the high heart rates among boys reflect the prodigality of youth, as contrasted with the conservatism of age, it is well established that youth excels in bursts of intense activity, but that the older man may be superior in sustained activity of more modest intensity

Body Size

In animals, it is well established that the heart rate tends to be slower as body size increases. It may be 700 per minute in a mature resting bat weighing 10 gm and less than 40 per minute in a mule. Other factors that regulate the heart rate in healthy man are so predominant that, in man, a clear-cut relation between body size and heart rate is not evident.

Emotional State

Our studies in the Fatigue Laboratory generally were carried out on subjects who had participated so frequently that there was

little anticipatory rise in heart rate. On one occasion we investigated the performance of 10 athletic teen-age boys who had had no previous experience in the laboratory.¹⁹ Their heart rates are shown in Table 7-3.

TABLE 7-3 HEART RATES OF TEEN-AGE BOYS BEFORE AND DURING MAXIMAL WORK

	Maximum	Minimum	Mean
1.5 min before starting	106	70	95
0.1 min before starting	133	195	115
0.1 min after starting	150	115	136
0.5 min after starting	155	175	166
1.25 min after starting	193	177	184
2.25 min after starting	202	182	190

The mechanism of regulation is revealed partially by observations on dogs before and after sympathectomy. In a collaborative study, Brouha, Cannon, and I¹¹ demonstrated that, even after complete sympathectomy, dogs are capable of fighting, and of other activities such as running on a treadmill. Before sympathectomy, the average heart rate of our animals while standing quietly on the treadmill, before exercise, was about 110 beats per minute. The respiratory arrhythmia, always present to a certain degree, varied in any given animal from one experiment to another. If the dog's attention was attracted while he was waiting to run, the heart rate increased from 100 to 140 beats per minute. If the dog received any emotional stimulus, such as the signal that warned him that the treadmill was about to start, the heart rate would jump from about 120 to about 200 beats per minute (Fig 7-14), although the animal usually remained motionless. After sympathectomy the resting rate was under 80 beats per minute and the anticipatory rate attained following the signal was 115 to 140 beats per minute. In a grade of work that was easy for this dog, even after sympathectomy, the heart rate stabilized at 120 beats per minute during exercise; before sympathectomy the rate was 50 beats per minute higher (Fig 7-15). It was clear from these studies that, in easy work, the normal dog has a higher heart rate than is essential.

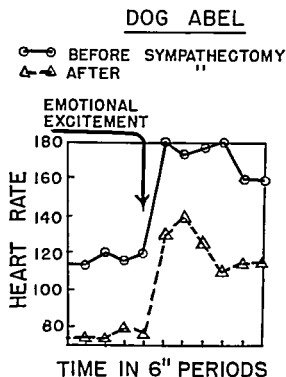


FIG 7-14 Effect of emotional excitement on the heart rate of dog, Abel, before and after sympathectomy (Courtesy *Journal of Physiology*.)

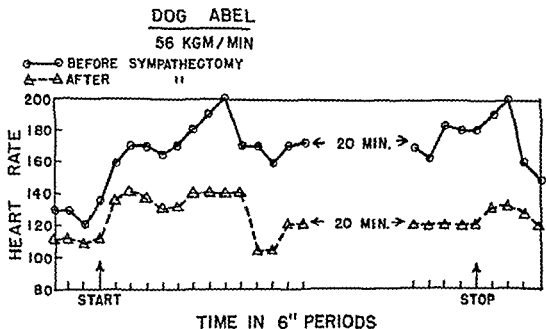


FIG 7-15 Heart rates before, during and after light work in dog, Abel, before and after sympathectomy (Courtesy *Journal of Physiology*)

CONCLUSION

There are many lessons to be learned from the facts marshaled above. One of the most obvious is that, in experiments that require observations on the heart rate, all factors that may influence its rate should be taken into account.

The term *steady state* should be used with caution. It is a relative term. A state that is approximately *steady* in a 10-minute study may be anything but *steady*, if the experiment lasts several hours. After all, we must stop to eat and to sleep. This is proof of cyclical changes in the internal environment.

BIBLIOGRAPHY

1. BOCK, A. V., VANCAUJAERT, C., DILL, D. B., FOLLING, A., and HURXTHAL, L. M. Studies in muscular activity, dynamical changes occurring in man at work, *J. Physiol.* 66: 136, 1928.
2. DILL, D. B., EDWARDS, H. T., and TALBOTT, J. H. Studies in muscular activity, factors limiting capacity for work, *J. Physiol.* 77: 49, 1932.
3. KEYS, A., BROZEK, J., HENSCHKE, A., MICHELSEN, O., and TAYLOR, H. L. "Circulation and Cardiac Function," v. 1, ch. 28

of *The Biology of Human Starvation*, Minneapolis, University of Minnesota Press, 1950.

4. FOLLING, M., EDWARDS, H. T., and DILL, D. B. Oxygen utilization in legs of normal men, effect of posture, *Am. J. Physiol.* 94: 459, 1930.
5. DILL, D. B. Economy of muscular exercise, *Physiol. Rev.* 16: 263, 1936.
6. DILL, D. B., EDWARDS, H. T., BAUER, P. S., and LEVENSON, E. J. Physical performance in relation to external temperature, *Arbeitsphysiol.* 4: 508, 1931.
7. DILL, D. B. Effects of physical strain and high altitudes on heart and circulation (Lewis A. Conner lecture), *Am. Heart J.* 23: 441, 1942.
8. SMITH, L. W., and FAY, T. Observations on human beings with cancer, maintained at reduced temperatures of 75°-90° Fahrenheit, *Am. J. Clin. Path.* 10: 1, 1940.
9. ROBINSON, S. Experimental studies of physical fitness in relation to age, *Arbeitsphysiol.* 10: 251, 1938.
10. DILL, D. B., and BROUHA, L. Etude sur le rythme pendant l'exercice ses rapports avec l'âge et l'entraînement, *Le Travail Humain* 5: 1, 1937.
11. BROUHA, L., CANNON, W. B., and DILL, D. B. Heart rate of sympathectomized dog in rest and exercise, *J. Physiol.* 87: 345, 1936.

CHAPTER 8 THE IMPORTANCE OF THE SIZE AND SHAPE OF THE HEART

A. C. Burton

In 1892, Woods,¹ a surgeon of Dublin, showed that the law of Laplace,² relating the tension in a stretched *membrane*, its shape, and the difference of pressure across the membrane, applied to the human heart:

$$P = T \left(\frac{1}{R_1} + \frac{1}{R_2} \right)$$

Woods estimated the radii of curvature at several points on the two ventricles and the thickness t of the ventricular wall at these points. He showed that the product

$$t \left(\frac{1}{R_1} + \frac{1}{R_2} \right)$$

was constant for the right ventricle, and also constant, but about six times greater, for the left. This is to be expected from the law of Laplace and the assumption that the force exerted by the muscle in the wall will be proportional to the thickness of the wall. From these measurements of the shape of the heart, it can be calculated that the ven-

tricular muscle must produce a force of about 5×10^5 dynes per square centimeter of cross section of the muscle. This agrees satisfactorily with what has been measured for various isolated muscles. Because of the law of Laplace, the ventricular wall must be much thicker at the places where it is *flatter* than where there is a great deal of curvature, e.g., at the apex.

The same law is found to apply to the arch of the aorta where on the *inside* of the arch the two radii of curvature are opposite in sign and subtract in the equation. On the outside of the arch the two curvatures sum. The wall is found to be correspondingly thicker on the inside of the arch.

The law of Laplace shows that when a heart is dilated, the tension that must be produced by the muscle, in order to produce the normal systolic pressure, is greatly increased. For twice the normal size it has to be at least four times as great. This is a very important consideration in heart failure

and accelerates greatly the reaching of the point of *decompensation* in Starling's law of the heart

Even though a dilated heart may be in the stage of compensation, and the increased force of contraction enables it to produce the same pressure in spite of its greater size, there will be a serious loss of cardiac efficiency. This is because the total cardiac load and requirement for oxygen depends not only on the work done in pumping blood, but also on the energy cost of maintaining tension in the heart muscle (*Tension* \times *Time* of A. V. Hill and co-workers³). This was pointed out as long ago as 1913 by Weiss⁴ and has been discussed by Wiggers⁵ and Katz,⁶ and more recently by Starr⁶ and Burch.⁷ If the heart is larger, the tension developed even to do the same amount of work must be greater, and therefore the oxygen required increases. Thus the efficiency of the heart depends upon its size

The classical researches of Starling and Visscher⁸ and the more recent work of Visscher⁹ showed that the oxygen requirement of the heart is determined by its diastolic size and is relatively independent of the work it has to do. This is explicable in terms of the law of Laplace, and indicates that, of the two terms in the total energy turnover, that depending on (*tension* \times *time*) is more important than that depending on the mechanical work. Preoccupation with the details of calculation of the work of the heart should not make us forget that this is much the smaller item in the total load of the heart. The serious consequences of an increase in heart size are emphasized when the classical laws of physics are applied to cardiac func-

tion.¹⁰ It is also pointed out that the clinical use of heart rate rather than cardiac work as an index of stress on the heart is completely justified. An increase in heart rate increases the major factors of (*tension* \times *time*) as well as the minor factor of external work, and is probably much better than the work alone as an indication of total energy requirements

BIBLIOGRAPHY

1. WOODS, R. H. A few applications of a physical theorem to membranes in the human body in a state of tension, *J. Anat. & Physiol.* 26:362, 1892.
2. LAPLACE, P. S. DE. *Mecanique celeste* 5 vols., 1799-1825.
3. HARTREE, W., and HILL, A. V. The regulation of the supply of energy in muscular contraction, *J. Physiol.* 55:133, 1921.
4. WEISS, G. Le travail du coeur, *J. Physiol. et Path. gen.* 15:999, 1913.
5. WIGGERS, C. J., and KATZ, L. N. Static and dynamic effort of the heart during ejection, *Am. J. Physiol.* 85:229, 1928.
6. STARR, I. Units for the expression of both static and dynamic work in similar terms and their application to weight lifting experiments, *J. Appl. Physiol.* 4:21, 1951.
7. BURCH, G. E. The dilated heart, *AMA Arch. Int. Med.* 96:571, 1955.
8. STARLING, E. H., and VISSCHER, M. B. Regulation of the energy output of the heart, *J. Physiol.* 62:243, 1927.
9. VISSCHER, M. B. Physiological principles of importance in heart failure and its treatment, *Journal-Lancet* 57:309, 1937.
10. BURTON, A. C. The importance of the shape and size of the heart, *Am. Heart J.* 54:801, 1957.

CHAPTER 9 CARDIAC FUNCTION IN CONGESTIVE HEART FAILURE IN MAN

Ludwig W. Eichna

A major concern of the clinical investigator is the extent to which the concepts of basic physiology, based as they are on measurements on excised organs, parts of organs, "intact" but anesthetized and manipulated animals, are applicable to man. This presentation will be concerned with cardiac function in congestive heart failure in man, a disease state generally considered to represent the prototype of failure of the heart muscle. An attempt will be made to indicate areas wherein the clinical physiologist utilizes, discards, or must modify the concepts of myocardial function as developed by basic physiology.

Three points have been chosen for consideration: (1) recognition of congestive heart failure and its differentiation from non-cardiac circulatory congestion, (2) reliability of presently used hemodynamic cardiovascular measurements as indexes of competency of myocardial function in congestive heart failure and after recovery therefrom,

and (3) applicability of Starling's law of the heart to man with normal circulation and with congestive heart failure.

RECOGNITION OF CONGESTIVE HEART FAILURE

Strange as it may at first seem, a primary problem of the clinical investigator is to define and recognize congestive heart failure. Such a definition is necessary not only for the sake of clarity in its own right, but is mandatory if the clinical investigator and basic physiologist are to consider the same problem and permit pertinent data and concepts to be interchanged.

It is generally agreed that congestive heart failure is a disease syndrome that results from chronic myocardial failure, that is, failure of the heart muscle cell. The diagnosis of congestive heart failure has traditionally been made, clinically, by demonstrating circulatory, that is, venous, congestion behind

either the right ventricle, manifested by distended neck veins, hepatomegaly, and edema, or behind the left ventricle, indicated by dyspnea, orthopnea, and rales, or behind both ventricles. Only the manifestations of such circulatory congestion are evident, not their cause. Clinicopathologic correlation long ago established that this circulatory congestion occurred in patients with diseased hearts and more recent hemodynamic measurements have indicated that the cardiac output in these cardiac patients is less than normal. The clinician has, therefore, come to consider all circulatory congestion as due to heart failure and specifically to failure of the myocardium to propel adequate amounts of blood. Is such a consideration valid? Studies on patients presenting with circulatory congestion indicate that both systemic and pulmonary vascular congestion are nonspecific hemodynamic manifestations and that such congestion may arise on a noncardiac basis to produce a disease state, which, clinically, simulates congestive heart failure arising from failure of the heart muscle as a pump.

The following situation will illustrate such noncardiac circulatory congestion simulating congestive heart failure. Consider the result of overadministration of fluid intravenously to a patient with a normal heart, particularly when the fluid is hypertonic or the urinary output is decreased. (The normal heart is important to the argument.) The blood volume is increased, vascular congestion appears, and the nonspecific signs and symptoms of circulatory congestion develop. Clinically, the manifestations are identical with those of congestive heart failure. But has the heart failed? It is difficult to understand how the heart by improving any of its functions, including increasing the cardiac output, can relieve the circulatory congestion.

Studies on patients presenting with circulatory congestion and a diagnosis of congestive heart failure have led to the separation of these patients into two major types, those in whom the circulatory congestion is due to myocardial failure and those in whom the circulatory congestion is noncardiac in origin. The data permit the noncardiac circulatory

congestions to be divided into three categories. Category I is characterized by mechanical obstruction to blood flow in and about the heart. A typical example is severe mitral stenosis of the "tight" or "pinch-cock" type, where the heart is not enlarged and right ventricular failure is absent. Tricuspid stenosis and constrictive pericarditis are other examples. Category II is characterized by excessive accumulation of water and salt in patients without heart disease and includes the edematous states associated with anuria of lower nephron nephrosis, with excessive administration of the salt-retaining substances ACTH, DCA, and cortisone and with acute glomerulonephritis. Category III contains the hyperkinetic circulatory congestions, the so-called high output heart failure, and includes the edematous states associated with beriberi, arteriovenous fistula, and anemia, again in patients without heart disease. In contrast to these noncardiac circulatory congestions are the typical congestive heart failures in cardiac patients with hypertension, coronary arteriosclerosis, and valvular heart disease, all with marked cardiac enlargement.

If these three types of circulatory congestion are of noncardiac origin, they should differ from congestive heart failure owing to myocardial failure in a number of significant hemodynamic functions. Accordingly, hemodynamic differences were sought and found in the following three functions: (1) cardiac dynamics and hemodynamics of the circulation as a whole, (2) the circulation and function of individual organs, in this instance the kidney, and (3) the hemodynamic and clinical responses to specific medication.

The data were obtained by simultaneous determination of all functions by standard methods based on right heart catheterization, the Fick principle, direct recording of vascular pressures, and renal clearances.³ These methods apply to all data presented throughout the paper.

Cardiac and Total Circulatory Dynamics

The intracardiac pressures, right atrial and systolic and diastolic pressure in the pulmonary artery and right ventricle, were ele-

vated in all three categories of noncardiac circulatory congestion to a degree comparable to the elevations in congestive heart failure. The sole exception was the normal end diastolic pressure in the right ventricle in mitral stenosis, an expected finding since the right ventricle had not failed. The comparable elevation of intracardiac pressures in the noncardiac circulatory congestions and in congestive heart failure is simply an indication of the similarity in location and degree of the circulatory congestion in the two states. The remaining data will examine whether the two major types of circulatory congestion were associated with the same or different hemodynamic causes.

With respect to blood volume, in patients with congestive heart failure total blood volume was increased above the predicted normal, whereas in the noncardiac circulatory congestions the blood volume often was normal (mitral stenosis, lower nephron nephrosis, acute nephritis) or when increased (cortisone therapy, beriberi, anemia) the increases were quantitatively less marked than in congestive heart failure. Here, then, is a distinction between the two types of circulatory congestion. The occurrence of increased intracardiac pressures in the presence of normal or low blood volumes presents, at first glance, a paradox. A possible explanation is that vasoconstriction effected a redistribution of blood from the venule and capillary beds to the more distensible central collecting compartments, the large veins and atria. Furthermore, this increased local blood volume is then subjected to increased venous and cardiac tone, for vasoconstriction affects all parts of the vascular tree, including the veins and cardiac chambers.

When the more primary function of cardiac output is considered, the differences between congestive heart failure and the three categories of noncardiac circulatory congestion become more evident and more fundamental. In congestive heart failure the cardiac index was typically below normal; in contrast the cardiac index was essentially normal in mitral stenosis, cortisone-induced edema, and acute nephritis, and the values

were high in anemia and beriberi. Furthermore, the arterial-mixed venous oxygen difference, relating blood supply (cardiac output) to the demand for it (oxygen consumption), was typically elevated in congestive heart failure in sharp contrast to the normal values in categories I and II and the low values in beriberi and anemia.

Renal Hemodynamics

Significant differences between the three categories of noncardiac circulatory congestion and congestive heart failure were indicated by differences not only in the dynamics of the circulation as a whole but also in the circulation and functions of an individual organ, the kidney. In congestive heart failure renal plasma flow was markedly reduced and glomerular filtration rate was moderately decreased, resulting in the strikingly increased filtration fraction, so characteristic of this type of circulatory congestion.² In contrast, in all three categories of noncardiac circulatory congestion renal plasma flow and glomerular filtration were only slightly reduced or normal (supernormal in the congested state induced by ACTH) and, of particular significance, the filtration fraction was not increased. Glomerulonephritis is excluded from this consideration because of the intrinsic renal disease and the oliguria of the patient with lower nephron nephrosis did not permit accurate measurements of renal clearance.

Effect of Digitalis

Digitalis is generally considered to have a direct effect on heart muscle and by this action to return the abnormal hemodynamic functions of congestive heart failure to, or well toward, normal. This effect has been considered to be quite specific for heart muscle failure. Accordingly, it was critical to determine whether digitalis had a similar hemodynamic effect in noncardiac circulatory congestion. Single therapeutic doses (1.0 mg to 1.5 mg) of digoxin were administered intravenously and were observed to produce no or minimal hemodynamic effects in the three categories of noncardiac congestion, in contrast to decided

improvement in hemodynamic function in congestive heart failure. Thus, in congestive heart failure, following intravenous administration of digoxin, the subnormal cardiac output rose to normal values, the abnormally high A-V oxygen difference decreased to normal levels, the elevated residual intracardiac pressure (right atrial and right ventricular end diastolic) fell markedly, but not to normal, and a brisk diuresis of electrolytes and water ensued.² These changes occurred concomitantly, began 15 minutes after injection of the drug, and reached their maximum in 90 to 120 minutes. In contrast, in all three categories of noncardiac circulatory congestion the cardiac output and A-V oxygen difference remained unchanged, the elevated residual intracardiac pressures remained unaltered, or fell only slightly and much less than in congestive heart failure, and water and electrolyte diuresis were absent or at best minimal. Finally, when the patients were fully digitalized with digitalis leaf given orally, the clinical results paralleled the acute effects of digoxin given intravenously, decided improvement in congestive heart failure, little or no clinical benefit in the three noncardiac congested states.

Follow-Up Observations

Long term follow-up observations also indicated differences in the basic nature of the two major types of circulatory congestion. When the heart muscle is involved the patient, in spite of the digitalis and diuretic therapy, lapses repeatedly and with shorter intervals of compensation into chronic congestive heart failure. In this state the patient dies, usually within 5 years of the onset of the first circulatory congestion. In the noncardiac circulatory congestions treatment of the primary, extracardiac cause produces a diuresis and relief from the circulatory congestion. The relief is permanent and without subsequent relapse or symptoms. Thus, mitral commissurotomy, pericardial decortication for constrictive pericarditis, withdrawal of salt-retaining hormones, spontaneous healing in acute nephritis, transfusion for the anemia, and vitamins for the beriberi have

all resulted in the appropriate instances in complete and permanent cure, as judged by follow-up observations over periods of 3 to 7 years in subjects receiving no cardiac or diuretic therapy. It would be difficult to consider that the heart was involved in such circumstances.

The differences in the general and local hemodynamic functions, in the response to specific medication and in the long-term course between congestive heart failure and the three categories of noncardiac circulatory congestion make it difficult to accept for all states of circulatory congestion the common etiology of heart muscle failure. Since the states designated noncardiac circulatory congestion occurred in subjects without preceding or subsequent heart disease, without impairment of specific cardiac functions and without hemodynamic response to digitalis, it seems reasonable to consider that these circulatory congestions developed in the absence of heart, that is, myocardial, failure. The recognition of noncardiac circulatory congestion is based on hemodynamic differences from congestive heart failure, and such a differentiation seems more tenable than grouping all circulatory congestions under the single category of congestive heart failure, simply because of the occurrence in all instances of the nonspecific clinical manifestation of circulatory congestion.

The following classification of circulatory congestion is suggested.

Circulatory (Venous) Congestion

(Increased local blood volume plus increased vascular tonus)

Cardiac

Congestive heart failure—low cardiac output

Noncardiac

Intracardiac obstruction—normal cardiac output

Water and salt retention—normal cardiac output

Hyperkinetic congested states—high cardiac output

This classification holds that circulatory congestion, both systemic and pulmonic, is a nonspecific hemodynamic manifestation that results whenever local, that is, venous blood volume is increased and under the influence of increased vascular tone. Such congestion may occur when the heart is diseased and the myocardium fails. This is congestive heart failure and should be differentiated from similar circulatory congestions occurring in three types of circumstances, namely, intracardiac obstruction to blood flow, excessive retention of salt and water, and hyperkinetic states, in all of which the heart appears not to fail as a pump. It is suggested that the term "congestive heart failure" be applied to the circulatory congestion that results from primary myocardial insufficiency and hence failure of the heart's propulsive force, and that this state be differentiated from circulatory congestions, which appear not to be of cardiac origin, for these states the term "noncardiac circulatory congestion" is proposed.

Other authors have suggested that not all circulatory congestions are due to failure of the heart as a pump⁴ and that noncardiac factors may be responsible for the circulatory congestions associated with acute nephritis,⁵ ACTH and cortisone therapy,⁶ massive intravenous infusions,⁷ beriberi and arteriovenous fistula.⁸

HEMODYNAMIC MEASUREMENTS AS INDEXES OF MYOCARDIAL FUNCTION

Having indicated the primacy of measurements of hemodynamic function in the recognition of the myocardial failure that results in congestive heart failure, it is pertinent to examine to what extent such measurements perform their intended function. Three points will be considered: (a) relationship between hemodynamic function and severity of congestive heart failure, (b) follow-up observations relating hemodynamic function with length of survival following recovery from congestive heart failure, and (c) impaired hemodynamic function without associated congestive heart failure.

Hemodynamic Function and Severity of Congestive Heart Failure

At the time of the patient's first congestive heart failure, the severity of the clinical manifestations of cardiac failure generally paralleled well the degree of abnormality of cardiac function, as determined by cardiac output and arterial-mixed venous oxygen difference. Full clinical recovery from congestive heart failure by digitalis medication was usually associated with considerable improvement toward normal in cardiac function (cardiac output and A-V oxygen difference) and in residual intracardiac pressures.⁹ The improvement in cardiac function was often not to normal values and right ventricular systolic pressure, and pulmonary artery pressures often remained elevated, whereas the residual intracardiac pressures were returned to normal in all cases. In this phase of recovery, subjects remained active and capable of considerable work without symptoms of abnormal cardiac function as long as they continued their digitalis medication. Indeed, this phase has been observed up to one and one-half years in some patients in spite of lapse from digitalis medication and hence for many months when digitalis was probably no longer in the body. This state of recovery from congestive heart failure represents asymptomatic failure of normal cardiac function and is not recognized by clinical criteria.

Inevitably, patients with heart disease and myocardial failure lapse into chronic congestive heart failure. Vigorous diuretic therapy and salt restriction usually relieve the clinical symptoms and signs. Such improvement is associated with fall of residual intracardiac pressures (right atrial, right ventricular end diastolic) to normal levels, an indication that the circulatory congestion has been relieved; in contrast the indexes of cardiac function (cardiac output and A-V oxygen difference) remain markedly abnormal and change either not at all or improve very little at best.

These observations indicate that the clinical yardsticks for the recognition and evaluation of reduced cardiac function are ex-

tremely gross and at best are capable of recognizing failing cardiac function only when the impairment is so great that cardiac function is near the last stages of maintaining circulation. Furthermore, present clinical criteria recognize not cardiac function but rather circulatory congestion, and the two need not be related. Finally, it is the circulatory congestion, not the reduced cardiac output, which is the cause of distressing symptoms to the patient and incapacitates him. The reduced cardiac output may force upon the patient lessened activity but is not in itself symptom-producing.

Hemodynamic Function and Duration of Survival After Recovery from Congestive Heart Failure

In an attempt to assess the reliability of the hemodynamic measurements of cardiac function employed by the clinical physiologist, the relationship between these measurements made after recovery from congestive heart failure and the subsequent duration of life was determined. The subsequent length of life may be considered to be related to the severity of the cardiac involvement: the more severely diseased the heart muscle, the more rapid the subsequent lapses into heart failure and the shorter the ultimate duration of life. Since patients seek medical care with varying degrees of severity of congestive heart failure, measurements made during such failure could not be used. Accordingly, measurements made after full digitalization and recovery from congestive heart failure were selected as indicating the degree of impairment of cardiac function. Such measurements obtained after recovery from the first congestive heart failure were compared for subjects who then survived 5 or more years with subjects who died within 5 years of their first congestive failure.

Determinations were available on 9 subjects who had survived for 5 or more years (three subjects for 5 years, three for 6 years, and three for 7 years). For comparison, the data for subjects known to have died within 5 years of their first failure were derived

from the same years of study as the 5-year survivors. In this second group were 10 subjects who lived for periods varying from three months to two years and eleven months. The ages of the subjects and types of heart disease in the two groups were comparable.

The data indicated no differences between the two groups with respect to the following cardiac and hemodynamic measurements: cardiac index, A-V oxygen differences, total body oxygen consumption, systemic arterial pressure, residual intracardiac pressure in the right atrium, and end of diastole in the right ventricle, or blood volume. In two respects the 5-year survivor group appeared to have suggestively less abnormal function: (1) their right ventricular systolic pressure and pulmonary artery pressures were perhaps not as abnormally high, and (2) on exercise they were more capable of elevating cardiac output, at least to some extent, whereas the nonsurvivor group satisfied the increased oxygen requirement of exercise almost entirely by increased oxygen extraction from blood.

This analysis suggests, contrary to original expectation, that the shorter duration of life was not associated with distinctly greater abnormality in cardiac and hemodynamic function. The analysis is incomplete and the number of subjects too few; nevertheless, it suggests that the hemodynamic measurements used by the clinical physiologist to assess cardiac function are at best very gross yardsticks. The data obtained can indicate whether cardiac function is abnormal or normal; they appear unable to differentiate satisfactorily between degrees of abnormality.

Impaired Hemodynamic Function Without Congestive Heart Failure

The occurrence of impaired cardiac and hemodynamic function in subjects without impairment of activity or development of congestive heart failure indicates further the inadequacy of present hemodynamic measurements to assess cardiac function. This situation has usually been observed in elderly

subjects, who may have cardiac indexes varying between 1.60 and 2.25 liters/sq m / min. without symptoms or evidence of a disease state. For example, a man aged 71 had in 1950 a cardiac index of 1.84 liters/sq.m / min , in 1952, 1.70 liters/sq m / min , in 1955, 1.75 liters/sq m / min , and now in 1957 remains well and for some reason enjoys physical exertion and boastfully tells of walking up 5 flights of stairs without difficulty and walking 50 city blocks for pleasure. Aging is associated with a progressive decrease in cardiac output, presumably in response to the lessened activity of the aged, indicated by their reduced total body oxygen consumption. As a result the A-V oxygen difference remains within normal limits. Under such circumstances cardiac output meets the bodily demands for blood, indicated by the normal A-V oxygen difference, and the subject remains asymptomatic. However, this is not always the case in these subjects and cardiac output may not meet the bodily demands, indicated by a rise in A-V oxygen difference, and still the subject remains symptom-free. Thus, the man previously mentioned had the following A-V O_2 differences in vols. per cent: 6.53 in 1950, 6.10 in 1952, and 6.59 in 1955. Apparently, then, increased extraction from blood is a satisfactory compensatory mechanism for decreased cardiac output. If the extraction is not marked, bodily compensation is not accompanied by dysfunction of other organs and circulatory congestion with symptoms of congestive failure does not develop. When the extraction becomes marked, then organ function becomes grossly abnormal with subsequent development of circulatory congestion and a disease state.

All the foregoing data and considerations therefrom indicate the limitations of present hemodynamic measurements in determining the state of cardiac function in man. Gross as these measurements are, they are nevertheless superior to clinical manifestations that are nonspecific and need not relate to heart failure at all.

STARLING'S LAW OF THE HEART AND CONGESTIVE HEART FAILURE IN MAN

Starling's law of the heart⁹ has come to dominate present concepts of both normal and abnormal cardiac function and has been extrapolated to apply to heart failure in man. It is still a pertinent question to what extent are observations and concepts—based on acute experiments on excised organs¹⁰ or even anesthetized, partially intact animals,¹¹ both preparations usually devoid of normal humoral and neurogenic influences, made to "fail" acutely by extreme and artificial means, in an unsteady state and of species not subject to spontaneous heart muscle failure—applicable to congestive heart failure in man, a spontaneously occurring but steady state of circulatory abnormality, still responsive to neurogenic and humoral influences, and due to chronic disease of heart muscle. Attempts, utilizing large infusions to alter ventricular filling pressure, have been made to determine whether Starling's law applies to congestive heart failure in man.¹² The large changes in blood volume induced in these attempts and in experiments on intact animals present an undesirable feature since the effect of volume change alone on cardiac output could never be determined.

The advent of vasodilator drugs offered the possibility of reducing ventricular filling pressure by noncardiac means and without alteration of blood volume, permitting thereby a test of the applicability of Starling's observations to man with both normal circulation and congestive heart failure. The drug Arfonad (trimethaphan camphorsulfonate) was chosen to lower vascular pressures because it has no direct myocardial or cardiac action and produces its effects by blocking autonomic ganglia, both sympathetic and parasympathetic. All functions were measured simultaneously. After 3 sets of control determinations, vascular pressures were lowered to and maintained at desired levels by a pump-controlled infusion of Arfonad (usually 0.4 to 1.2 mg/min). All measurements were repeated after the low-

ered pressures had been maintained for 15, 30, and 60 minutes. The Arfonad infusion was then stopped and the pressures were allowed to return to the initial levels, when the final measurements were made.

The original hope that intracardiac filling pressure could be lowered without reduction in systemic arterial pressure was not attained. Therefore, the fall in pressure was carefully controlled. In hypertensive patients, systemic arterial pressure was lowered to levels slightly above normal, in normotensive patients systolic arterial pressure was maintained between 90 and 100 mm Hg. Severe hypotension and circulatory collapse were avoided.

The hemodynamic effects associated with the lowered pressures were qualitatively similar in subjects with similar circulatory states, permitting thereby pooling of the subjects and analysis of their data in three groups: Group I, 10 subjects, 5 normotensive and 5 hypertensive, with typical low-output congestive heart failure, Group II, 4 subjects, 2 normotensive and 2 hypertensive, recovered from typical congestive heart failure by digitalis and without signs or symptoms of heart failure but with cardiac output below normal, Group III, 5 subjects, 2 noncardiac subjects convalescent from pneumonia and alcoholism, respectively, and 3 cardiac patients, 1 normotensive and 2 hypertensive, fully compensated from congestive heart failure by digitalis and without signs or symptoms of heart failure and with normal cardiac output.

In all three groups of subjects, pressures in the systemic artery, pulmonary artery, right ventricle during systole and the filling pressures indicated by right atrial pressure, right ventricular and diastolic pressure, and pulmonary "wedge pressure" fell during the ganglionic blockade, remained at the desired level during the infusion of Arfonad, and returned to control levels following cessation of the infusion. These measurements indicate the similarity of the changes in vascular pressures in the three groups of subjects.

While the vascular pressures were at their

lowered level, cardiac output increased slightly but definitely (10 to 20 per cent and not to normal levels) in both the normotensive and hypertensive subjects with congestive heart failure (Group I). In contrast, cardiac output fell (up to 20 per cent and often below the normal range) in the compensated cardiac patients and normal subjects (Group III). Arterial-mixed venous oxygen difference fell toward normal, but usually not to normal, in both the normotensive and hypertensive subjects in congestive heart failure (Group I). Again in contrast, the A-V oxygen difference increased slightly or remained unchanged in the compensated cardiac and noncardiac subjects (Group III).

The responses in the compensated cardiac subjects with low cardiac output (Group II) were somewhat variable and intermediate between the effects noted in congestive heart failure and the fully compensated cardiac subjects.

The hemodynamic responses during the lowered vascular pressures may be considered consistent with Starling's law, at least in that interpretation which relates cardiac output to intracardiac filling pressure. Thus cardiac function improved, as measured by cardiac output and A-V oxygen difference, as the high filling pressures of congestive heart failure were lowered. The reverse, a deterioration of function, occurred when the normal ventricular filling pressures of noncardiac and compensated cardiac patients were lowered. However, there was the disconcerting observation that fairly often the increase in cardiac output, so apparent when the elevated filling pressures were first lowered, tended not to persist and the increase in cardiac output at times gradually decreased even though the filling pressures remained lowered.

When Starling's law is considered in its more correct terms of cardiac work, then cardiac work *did not increase* when the elevated intracardiac filling pressures of congestive heart failure were lowered. In both the normotensive and hypertensive patients with congestive heart failure,

minute work index and stroke work index of both the right and left ventricles remained essentially unchanged, even though cardiac output increased, presumably in response to the fall in filling pressure. In the normotensive and hypertensive compensated cardiac patients and in the noncardiac subjects the expected decrease in cardiac work occurred; minute work index and stroke work index of both left and right ventricles decreased as filling pressures fell.

The interpretation of these changes in heart work in reference to Starling's law is rendered difficult, if not impossible, by the fall in arterial pressures that occurred in these observations. Thus, in the patients with congestive heart failure, the tendency for an increase in cardiac work as a result of the greater cardiac output was offset by the lowered arterial pressure and the work of the heart remained unchanged. Starling's law, accordingly, did not obtain in congestive heart failure with high ventricular filling pressures. The decrease in cardiac work when the normal filling pressures in compensated cardiac and noncardiac subjects were lowered is consistent with Starling's law, a decrease in cardiac work at the lower filling pressures. However, in these observations the lowered arterial pressures had a greater influence in the reduced cardiac work than the observed small fall in cardiac output.

The factual observation remains that primary vasodilatation in congestive heart failure seemed to result in at least a temporary increase in cardiac output and hence total body blood flow without an increase in work by the heart. Presumably, lowering the pressure against which the heart ejects blood permits an impaired myocardium to expel more blood without an intrinsic improvement in function of the heart muscle. Such a result may be of advantage to the circulation where a failing myocardium is not capable of improvement and greater work. However, no extrapolation of the acute results of the short period of vasodilatation of these studies to the effects of chronic vasodilatation can be made.

Since the experimental procedure lowered pressures on both sides of the ventricles, it was not possible to determine the effect of changes in intracardiac filling pressure alone, as a test of Starling's law would require. Accordingly, a test of the applicability of Starling's law in man was not achieved. Recent and still preliminary experiments, utilizing 0.2 gm of sodium nitrite administered orally to lower intracardiac filling pressure without fall in systemic arterial and pulmonic arterial pressure, seem to suggest that Starling's law applies in both congestive heart failure and in normal circulation. Cardiac output and minute and stroke work index have increased when the high right atrial and right ventricular end diastolic pressures of congestive heart failure have been lowered. In noncardiac subjects, lowering of the same pressures was accompanied by a decrease in cardiac output and in minute and stroke work index.

Many more observations in varying circulatory states in man are necessary before it is fruitful to consider in any detail whether Starling's law of the heart applies in intact man. It is not proposed to discuss here the evidence suggesting that the law does apply, does not apply, or applies in part and only in those situations where the heart muscle is failing.

SUMMARY

Three points evolve from this discussion.

1. Clinically congestive heart failure is recognized by signs and symptoms of circulatory (venous) congestion behind one or both ventricles. However, such circulatory congestion is a nonspecific hemodynamic manifestation and may be of noncardiac as well as cardiac origin. The differentiation depends upon hemodynamic measurements, indicating normal cardiac function in the circulatory congestions of noncardiac origin and impaired cardiac function when myocardial involvement is the cause. A distinction between the two types of circulatory congestion is necessary in order to avoid confusion in considerations dealing with

cardiac function in disease and in order to relate properly the concepts of basic physiology to disease states in man

2. Although hemodynamic measurements are superior to clinical manifestations in the evaluation of impaired cardiac function, these measurements are still gross indexes of cardiac efficacy and impairment. Thus, reduced cardiac output may exist without impairment of activity or development of congestive failure, degrees of impairment of cardiac function do not necessarily correlate with clinical manifestation of congestive heart failure, and hemodynamic measurements do not predict satisfactorily duration of survival of subjects with heart disease

3. Attempts were made to determine the applicability of Starling's law of the heart in man, both with normal circulation and with congestive heart failure. When ventricular filling pressures were lowered by means of vasodilating agents, there resulted an increase in cardiac output but no change in minute or stroke work in congestive heart failure and a reduction in cardiac output and minute and stroke work when the circulation was normal. In view of the fall in arterial pressure as well as filling pressure in these observations, it is not clear whether this type of experiment served as a test of Starling's law. However, in congestive heart failure the resulting increased blood flow occurred without increase in cardiac work, a situation that may be favorable to the circulation in those instances where impaired heart muscle is incapable of increased work

BIBLIOGRAPHY

- 1 Cournand, A., Riley, R. L., Bredt, E. S., Baldwin, E. de F., and Richards, D. W., Jr. Measurement of cardiac output in man using the technique of catheterization of the right auricle or ventricle, *J Clin Invest* 24: 106, 1915
- 2 Briggs, A. P., Powell, D. M., Hamilton,

CHAPTER 10 ENERGETICS OF THE HEART AND CORONARY FLOW*

Louis N. Katz

Recently, renewed interest has arisen in the regulation of the performance of the heart, its energy cost, and the manner in which the energy is supplied. Among others, this has been the concern of this department. We have utilized a method developed in the department by Rodbard, Graham, and Williams¹ (Fig 10-1) in which the right ventricle receives (and pumps) only coronary blood via the coronary sinus, accessory coronary veins, and the Thebesian channels (constituting about 95 per cent of the coronary flow). This is metered. The blood from the systemic circuit (other than the coronary flow) is diverted from the right heart and is delivered by an artificial pump to the left heart through the lungs (together with the coronary blood coming from the right heart). The output of the left ventricle can thus be set at any desired level by the input

pump, which is metered. The resistance against which the left (and right) ventricle pumps can also be adjusted by a resistance on the aorta (and pulmonary artery). In this preparation, the influence of nerves and hormones can operate intact. It is a compromise between an entirely intact animal and a preparation approaching this while still permitting adequate measurements of the cardiac work, its oxygen consumption, and its coronary flow.*

In analyzing our data in correlation with earlier work of the department and that in the literature, it soon became apparent that a number of factors control the performance of the heart (Fig 10-2):

1. *The magnitude of its work-load*. Cardiac oxygen consumption tends to increase as cardiac work increases, up to a point.²

* In the studies with this preparation, I have had the co-operation of Drs A. Alella, H. Feinberg, A. Gerola, A. M. Katz, and D. Laurent, and the help of Mrs C. Bolene-Williams and Messrs F. L. Williams and E. Boyd.

* This work was supported by grants from the American Heart Association, the National Heart Institute, and by the Michael Reese Research Foundation.

FIG. 10-1. Diagram of the method used recently for the study of coronary flow and myocardial oxygen consumption (Fig 1 of Alella *et al.*⁹). The blood reservoir that receives vena caval blood is kept at body temperature, and the blood in it is pumped through a flowmeter to be aerated by the lungs. The azygos vein is tied. The right ventricle, deprived of vena caval and azygos inflow, pumps out only coronary sinus, accessory coronary venous, and right ventricular Thebesian drainage through a Starling resistance and a flowmeter to the reservoir. R.P.A. and L.P.A. are right and left pulmonary arteries respectively; R.P.V. and L.P.V. are right and left pulmonary veins, R.A. and L.A. are right and left atria, R.V. and L.V. are right and left ventricles, M.P.A. is the main pulmonary artery, C.A. are coronary arteries, C.S. is the coronary sinus, and T is a Thebesian drainage channel.

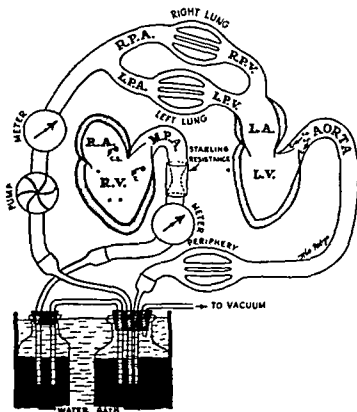
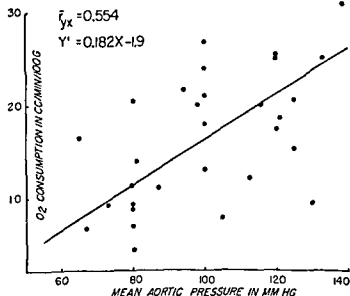


FIG 10-2. Relationship between myocardial oxygen consumption and mean aortic pressure (Fig 2 of Alella *et al.*⁹).



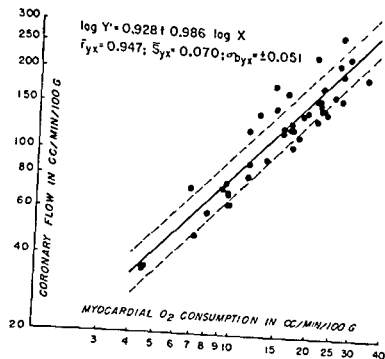


FIG 10-3 Logarithmic relationship between myocardial consumption and coronary flow (Fig 4 of Alella *et al*). Solid line is the regression line expressed by the equation. Dotted lines represent the standard error of estimate ($\pm S_{yx}$)

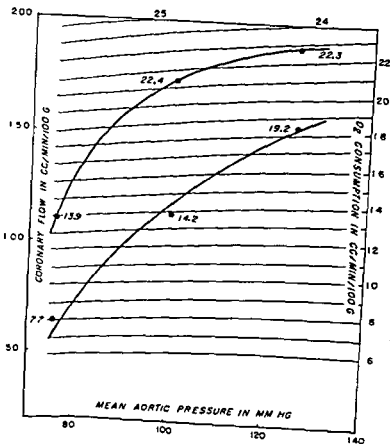


FIG 10-4 Nomogram relating cardiac oxygen consumption, coronary flow, and mean aortic pressure (Fig 5 from Alella *et al*). The curved lines connecting coronary flow and oxygen consumption (both in cc/min/100 gm heart weight) relate these two variables at different levels of coronary flow. The two lines rising sharply within intersecting curved lines represent actual experimental observations.

FIG 10-5 Graph showing the relationships between coronary flow and heart rate (upper curve) and between myocardial oxygen consumption/100 gm heart weight and heart rate (lower curve) (Fig. 1 from Laurent *et al**)

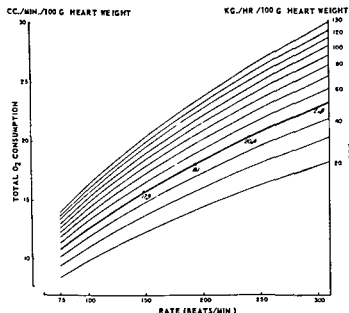
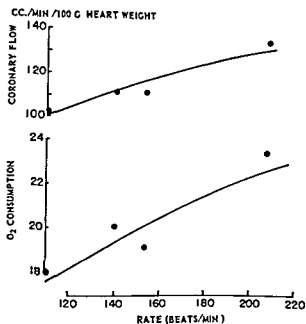


FIG 10-6 Nomogram of cardiac isowork lines relating myocardial oxygen consumption to heart rate (Fig 2 from Laurent *et al**) The heavy line represents experimental points observed with work level of 50 kg/hr/100 gm heart weight

2 *The kind of work-load* Increase in cardiac inflow tends to increase cardiac oxygen consumption less than a like increment in cardiac work resulting from an increase in arterial pressure ^{3,4}

3 *The heart rate* For a given cardiac work-load an increase in heart rate per se leads to an augmentation of cardiac oxygen consumption ⁵

4 *The stress-adapting mechanism* (see below)

5 *Hypoxemia* (see below)

6 *Drug action* (see below).

Furthermore, it was apparent that heart performance, while determined by heart size (end-diastolic volume), is also determined by the nature of its contraction (after-load) and by specific actions on its contractile and

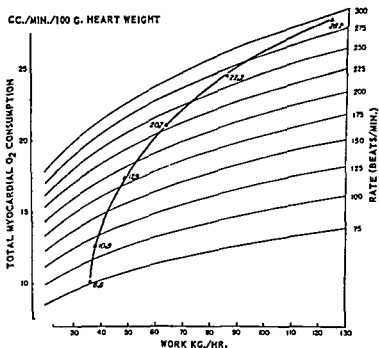
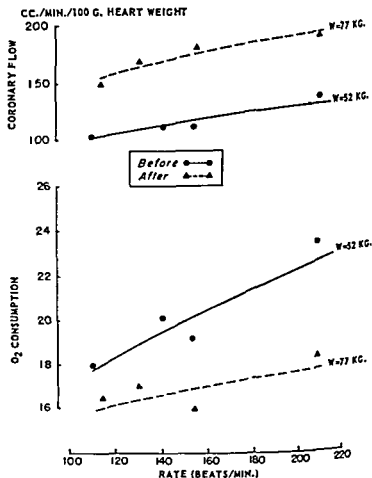


FIG. 10-7. Nomogram of isorate lines relating myocardial oxygen consumption to cardiac work, in kg/hr./100 gm heart weight (Fig 3 from Laurent *et al.*¹). The observed cardiac oxygen consumption values of two experiments have been plotted.

FIG 10-8 Graphs showing relationship between coronary flow and heart rate, and between cardiac oxygen consumption and heart rate before (solid lines) and after (dash lines) the occurrence of the *stress-adapting mechanism* (Fig 6 from Laurent *et al.*¹), initiated in this case by an increase in cardiac work from 52 to 77 kg/hr/100 gm heart weight at a heart rate of 207 beats/min



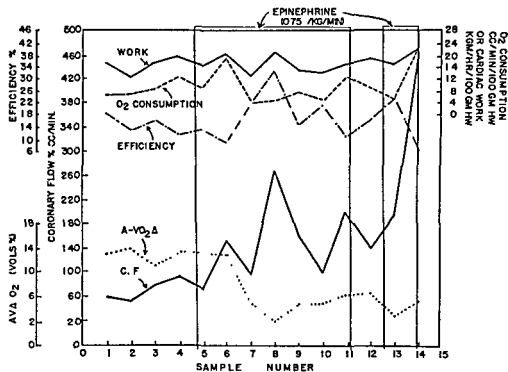


FIG 10-9 Graph showing data from an experiment in which the *stress-adapting mechanism* developed following exhibition of 1-epinephrine by constant infusion at the rate of 0.75 γ /kg/min. The decrease in cardiac oxygen consumption, with its associated increase in efficiency of the heart and in coronary flow simultaneous with a decrease in coronary A-V oxygen difference, is clear.

relaxing mechanisms independent of the end-diastolic volume and after-load^{2,3,4,6}. It was also apparent that the heart volume at the end of systole is large in the intact animal and can be called upon, together with that in the lungs and central veins, for immediate urgent needs^{3,4}. Furthermore, heart rate changes were found to be as unimportant a means of adjusting heart performance as stroke output⁷. Thus, there are a number of factors that allow adjustments of the heart's performance to its load—not just one.

Under ordinary circumstances, it was apparent that the energy utilization of the heart as indicated by its oxygen consumption is adjusted primarily by the coronary flow^{5,8,9,10} (Figs 10-3, 10-4). A close relationship was found to exist between coronary flow and cardiac oxygen consumption, such that coronary venous oxygen content and coronary arteriovenous oxygen difference remains fairly constant^{7,9}. Actually, the

relationship between cardiac oxygen consumption and coronary flow is best expressed by a straight line. Coronary flow does not depend on heart rate (Figs 10-5, 10-6, and 10-7), cardiac output, or systemic blood pressure per se to any great extent except insofar as they affect the cardiac oxygen consumption^{6,7,8,9}.

With these facts in mind, several adjustments to unusual circumstances deserve consideration. The first consists of the adjustment to excessive load. It was found that when the cardiac load (or heart rate) was increased excessively, a sharp deviation of the oxygen consumption and coronary flow occurred, as if there had been a *shift in gears* in the form of a stress-adapting mechanism^{2,5,7}. This shift consisted of the following changes: (a) a reduction in cardiac oxygen consumption, so that the ratio work/O₂ consumption of the heart declined, (b) an increase in coronary flow making the

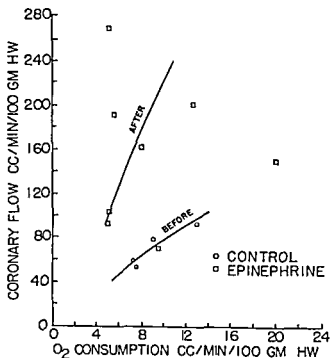


FIG 10-10. Graph based on data in Fig 10-9 showing the relationship of coronary flow to cardiac oxygen consumption before and after the development of the *stress-adapting mechanism*. Note the greater coronary flow for a given oxygen consumption after the *stress-adapting mechanism* appeared and the greater slope of the regression line.

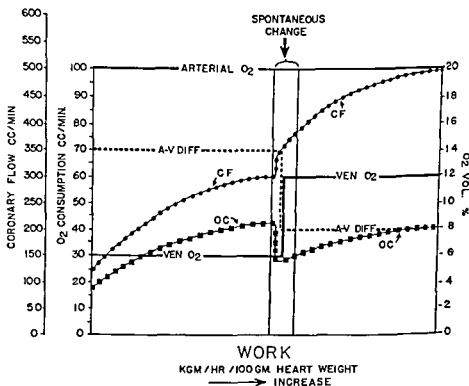


FIG. 10-11 Schematic presentation of the development of the *stress-adapting mechanism* as cardiac work increases, showing the simultaneous shift in cardiac oxygen consumption (OC), coronary flow (CF), coronary A-V oxygen difference (A-V Diff) and coronary venous oxygen content (VEN. O₂). ARTERIAL O₂ is the arterial oxygen content (Fig 12 of Katz').

FIG 10-12 Graph showing effect of hypoxemia upon cardiac oxygen consumption when cardiac work was between 40 and 80 kg/hr/100 gm. heart weight (Fig 8 from Katz *et al.*¹) Abscissa shows arterial oxygen content in volumes per cent.

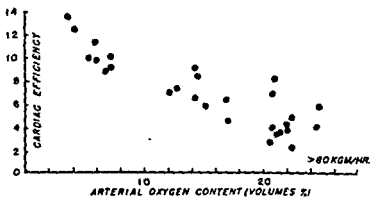
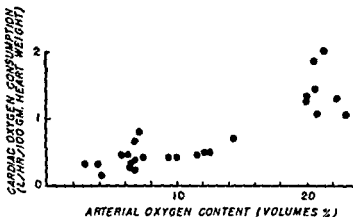
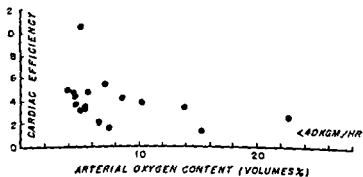
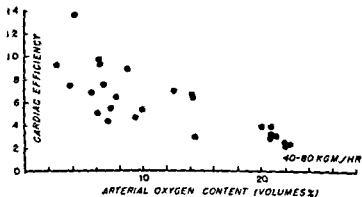


FIG 10-13 Graphs showing relationship between cardiac efficiency, that is, ratio cardiac work/cardiac oxygen consumption, and arterial oxygen content at three levels of cardiac work, as indicated in the lower right-hand corner of each individual graph (Fig 7 from Katz *et al.*¹)



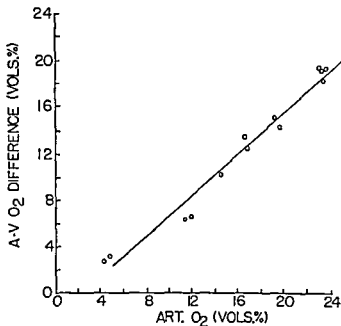


FIG 10-14. Graph showing effect of hypoxemia on coronary A-V oxygen difference. (Data recently obtained by Feinberg, Gerola, and Katz¹¹)

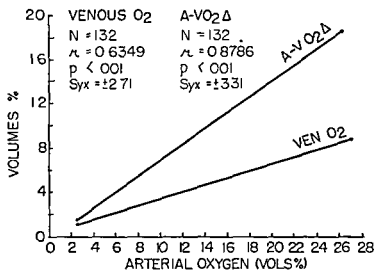


FIG 10-15 Graph showing effect of hypoxemia on coronary A-V O₂ difference (A-V O₂ Δ) and coronary venous O₂ content (Ven O₂) (Data recently obtained by Feinberg, Gerola, and Katz¹¹)

ratio coronary flow/cardiac oxygen consumption deviate from the one usually found; (c) an arterIALIZATION of coronary venous blood, and (d) a decrease in coronary arteriovenous oxygen difference. These changes were long-lasting (up to an hour and a half) and reversible. They are different, therefore, from those due to the oxygen debt phenomenon (Figs. 10-8, 10-9, 10-10, and 10-11). The increase in coronary flow provides an increase in the availability of oxygen (and other substrates) for the heart. Whether the heart in the stress-adapting

mechanism adjusts its internal processes of metabolism or simply obtains substrates from the blood *parasitically* without restoring them to their pre-existing condition is not yet determined; nor are the details by which the stress-adapting mechanism is brought about yet known.

The second unusual circumstance is hypoxemia. As the oxygen content of the arterial blood supplying the heart declines, coronary flow rises relative to the oxygen consumption of the heart,² and, at the same time, the slope of the regression curve relat-

FIG. 10-16 Graph showing different relationships between coronary flow and cardiac oxygen consumption at three levels of arterial O_2 content. \circ , 19-27 vol per cent, Δ , 7-18 vol. per cent, \square , 1-6 vol per cent. Note that for a given cardiac O_2 consumption the coronary flow is higher as hypoxemia develops. Further, the increment of coronary flow for each increment of cardiac O_2 consumption is greater as hypoxemia advances (Data recently obtained by Feinberg, Gerola, and Katz¹¹)

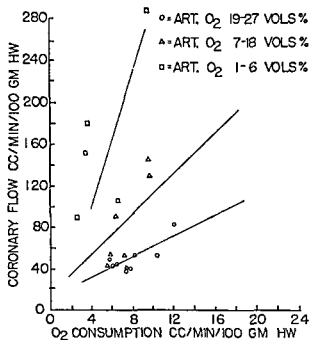
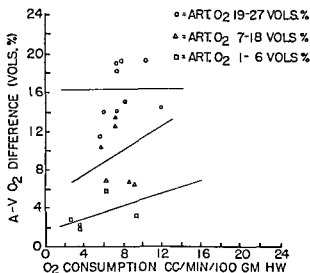


FIG. 10-17 Graph showing different relationships between coronary A-V O_2 differences and cardiac oxygen consumption at three levels of arterial O_2 content. Constructed as Fig. 10-16. Note that the level of A-V O_2 difference decreases (and the regression line increases in slope as hypoxemia progresses) (Data recently obtained by Feinberg, Gerola, and Katz¹¹)



ing the two also rises.¹¹ These reflect mechanisms that permit the availability of oxygen (and other substrates) to the heart to be kept up. At the same time, or soon thereafter, the extraction of oxygen increases to approach 90 per cent ultimately.¹¹ And, finally, the ratio cardiac work/cardiac O_2 consumption rises—the heart as it were is a *facultative anaerobe*^{2,11} (Figs. 10-12, 10-13, 10-14, 10-15, 10-16, 10-17, and 10-18). The mechanism by which these obviously protective changes are brought

about is not yet understood. Despite these protective mechanisms, further augmentation of hypoxemia ultimately leads to a deterioration of the heart's performance, and death ensues.

Finally, with the knowledge thus outlined we are in a better position to understand the manner by which drugs and other alterations affect the heart's performance and its coronary flow. This is important because it permits the distinction to be made between

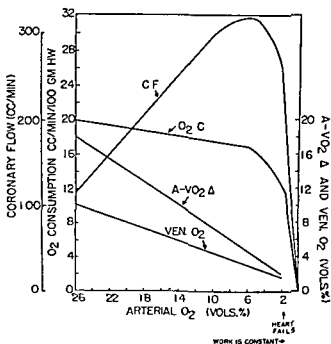
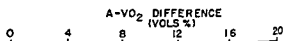


FIG. 10-18. Schematic presentation of the changes in cardiac oxygen consumption, coronary flow, coronary A-V oxygen difference, and coronary venous oxygen content as arterial oxygen content (abscissa) shifts from 20 volumes per cent down to 0. The work level is maintained constant, at the level set, until the available oxygen (coronary flow \times arterial oxygen) falls below a critical level. The work then declines progressively in an accelerating fashion as the arterial oxygen continues to fall. CF is coronary flow, $O_2 C$ is cardiac oxygen consumption, A-V $O_2 \Delta$ is coronary A-V oxygen difference and Ven O_2 is coronary venous oxygen content.



A = CONTROL

N = 10

$AVO_2 \Delta = 14.71$

$\sigma = \pm 2.84$

B = EPINEPHRINE

615/KG/MIN. (1-2.55)

N = 43

$AVO_2 \Delta = 13.33$

$\sigma = \pm 2.85$

C = EPINEPHRINE

2.08/KG/MIN (1.0-2.55)

N = 7

$AVO_2 \Delta = 11.86$

$\sigma = \pm 2.70$

A-B $t = 1.3680$ $p < .3$

A-C $t = 2.0300$ $p < .1$

A = CONTROL

N = 14

$AVO_2 \Delta = 15.04$

$\sigma = \pm 3.19$

B = NOREPINEPHRINE

435/KG/MIN (1-2.55)

N = 51

$AVO_2 \Delta = 12.37$

$\sigma = \pm 2.91$

C = NOREPINEPHRINE

135/KG/MIN (1.0-2.55)

N = 5

$AVO_2 \Delta = 11.96$

$\sigma = \pm 2.60$

A-B $t = 2.9790$ $p < .01$

A-C $t = 1.9300$ $p < .1$

FIG. 10-19 Chart showing effect of 1-epinephrine and 1-norepinephrine on coronary A-V O_2 difference. The horizontal bar shows the range and the vertical line the mean in each instance (Data recently obtained by Feinberg, Gerola, and Katz).

primary changes in coronary flow and those depending secondarily upon alterations in the myocardial oxygen consumption.

So far we have studied only the actions of the catecholamines.¹² Both 1-norepinephrine and 1-epinephrine shift the relationship of coronary flow to cardiac oxygen consumption—they have a primary action on the coronary vasculature. The coronary flow rises and coronary arteriovenous oxygen difference falls. The regression line relating coronary flow to cardiac oxygen consumption appears to be different for 1-nor-epinephrine than for 1-epinephrine (Fig. 10-19). Similar studies with other drugs should be equally informative.

Since this presentation was prepared in 1957, the field has advanced and our own work has expanded. Our views have therefore changed in certain respects. These later views have been summarized in published reviews.^{13,14}

BIBLIOGRAPHY*

1. RODBARD, S., GRAHAM, G. R., and WILLIAMS, F. L. Continuous and simultaneous measurement of total coronary flow, venous return and cardiac output in the dog, *J Appl Physiol* 6: 311, 1953.
2. KATZ, L. N., KATZ, A. M., and WILLIAMS, F. L. Metabolic adjustments to alterations of cardiac work in hypoxemia, *Am J Physiol* 181: 539, 1955.
3. KATZ, L. N. The sixty-ninth Mary Scott Newbold lecture: mechanism of cardiac failure, *Circulation* 10: 663, 1954.
4. KATZ, L. N. Symposium on regulation of performance of heart, analysis of several factors regulating performance of the heart, *Physiol Rev* 35: 91, 1955.
5. LAURENT, D., BOLENE-WILLIAMS, C., WILLIAMS, F. L., and KATZ, L. N. Effects of heart rate on coronary flow and cardiac oxygen consumption, *Am J Physiol* 185: 353, 1956.
6. KATZ, A. M., KATZ, L. N., and WILLIAMS, F. L. Registration of left ventricular volume curves in the dog with the systemic circulation intact, *Circulation Res* 3: 588, 1955.
7. KATZ, L. N. Design of proper experiments to investigate clinical angina pectoris and the importance of knowing the determinants of coronary flow in considering therapy of angina pectoris, *Ann New York Acad Sc* 64: 505, 1956.
8. KATZ, A. M., KATZ, L. N., and WILLIAMS, F. L. Regulation of coronary flow, *Am J Physiol* 180: 392, 1955.
9. ALELLA, A., WILLIAMS, F. L., BOLENE-WILLIAMS, C., and KATZ, L. N. Interrelation between cardiac oxygen consumption and coronary blood flow, *Am J Physiol* 183: 570, 1955.
10. ALELLA, A., WILLIAMS, F. L., BOLENE-WILLIAMS, C., and KATZ, L. N. Role of oxygen and exogenous glucose and lactic acid in the performance of the heart, *Am J Physiol* 185: 487, 1956.
11. FEINBERG, H., GEROLA, A., and KATZ, L. N. Effect of hypoxemia on coronary flow and cardiac oxygen consumption, *Fed Proc* 16: 36, 1957.
12. KATZ, L. N., WILLIAMS, F. L., LAURENT, D., BOLENE-WILLIAMS, C., and FEINBERG, H. Effects of 1-norepinephrine and 1-epinephrine on coronary flow and oxygen consumption of the intact open-chested dog, *Fed Proc* 15: 106, 1956.
13. KATZ, L. N. "The Factors Involved in the Regulation of the Heart's Performance," p. 70, in *Circulation (Proc Harvey Tercentenary Congress)*, Edited by J. McMichael, Oxford, England, Blackwell, 1958.
14. KATZ, L. N., and FEINBERG, H. The Relation of cardiac effort to myocardial oxygen consumption and coronary flow, *Circulation Res* 6: 656, 1958.

* Reference is made to recent work from this department. Earlier work of the department and that of others will be found in these references, especially 3 and 4.

REPORT OF PANEL ON BASIC PHYSIOLOGY

William H. Stewart

I will present a summary of the discussions of the panel on basic physiology when it met separately. The deliberations of the joint meetings with the clinical physiology panel will be reported by Dr. Burchell.

The basic mechanisms concerned with the function of the heart as a muscular machine for doing work were considered by the panel during the first two hours of its initial meeting. Dr. R. W. Ramsey outlined the historical development of our present-day knowledge of the thermodynamics of skeletal muscle. He pointed out that the once popular ideas of H. V. Hill and others, that muscle behaved in the manner of a stretched spring in which energy is stored in relaxation and liberated as work and heat in overcoming viscosity in contraction, were rendered untenable by such observations as the Fenn Effect. This latter is defined as the property of muscle that results in energy liberation being proportional to the work done in shortening plus a quantity independent of such work. He also noted that there is compelling evidence to indicate that relaxation is an active phenomenon and not

passive as would be required on the basis of the older hypotheses. Dr. Ramsey also pointed out that single muscle fibers do not fatigue in the usual sense. Their excitability may diminish but their contractility does not.

Possibly the most provocative of Dr. Ramsey's suggestions was that cardiac muscle is, perhaps, normally in the condition of what he has called the *delta state* of skeletal muscle, which occurs when the muscle is made to shorten to one third of its resting length. In the delta state muscle does not show active relaxation and probably does not show the Fenn Effect. Dr. Ramsey also pointed out that extremely little is known about the thermodynamics of heart muscle. The reasons for this are mostly technical. Finally, he said that, in contrast to skeletal muscle, where the force-velocity curve is, as he put it, chiefly an expression of the innervation pattern of that individual and not of the individual muscle, in the heart the force-velocity pattern depends on the ultimate contractile power of the tissue itself.

The second speaker was Prof. H. H.

Weber, who spoke on movements and contractile proteins of muscles and cells. He presented evidence for the view that the reaction between ATP and actomyosin is the fundamental one for motility in the entire animal kingdom. He pointed out that isolated muscle fibrils, extracted fiber models, and reconstituted contractile protein models as well as dead and extracted ameboid cells, spermatozoa tails, and fibroblasts all exhibit this characteristic motor response upon addition of ATP under suitable circumstances. He pointed out, furthermore, that sulfhydryl binding poisons have inhibitory effects of the same sort in each of these systems, and the effects are reversed by the addition of excesses of sulfhydryl compounds. He also pointed out that in some systems of a different nature ATP produces relaxation rather than contraction. In the discussion it was agreed that at the present time we are in great need of more information about the basic chemistry of contraction. Prof. Weber pointed out that recent x-ray and electron microscopic studies by the Huxleys have suggested the possibility that the contractile process may be accounted for by the sliding movement of the actin fibers alongside the L-myosin fibers in the so-called I-Band of the muscle. Dr. Ramsey pointed out that in any simple form, this theory would be inadequate because it would not account for the extent of shortening possible. However, Dr. Weber indicated that by a combination of folding and sliding it could account for the shortening.

The basic physiology panel then turned to more specific studies of the energetics of the heart itself. Dr. Sarnoff reported at length on the very beautiful studies made by him and his colleagues using what they call a *biochemically supported isolated heart preparation*. This preparation differs from the typical isolated heart in that the blood supplying it is passed in series during each cycle through an intact animal. This preparation was employed in an examination of the hemodynamic determinants of myocardial oxygen consumption. His most striking observation was that the most regular correlation between O_2 consumption and any other

variable measured was with the *Time Tension Index* (the product of mean systolic pressure \times the duration of systole). He pointed out that there is relatively very much less increased energy cost in doing increased cardiac work through increased cardiac output than against increased pressure. In fact, he found that there was no change in efficiency when work was increased by increasing pressure load, whereas efficiency increased greatly with increased cardiac output at constant arterial pressure. The reason for the correlation of O_2 consumption with the *Time Tension Index* is primarily in the increased duration of systole with increased cardiac outputs.

Dr. E. Braunwald then spoke on the hemodynamic effects of mitral and aortic regurgitation. He pointed out that the hemodynamic disadvantages were very much greater in aortic regurgitation than in mitral insufficiency. His quantitative evaluation involved measurements of LVEP (left ventricular end diastolic pressure) in relation to the left ventricular work at constant heart rates. He also pointed out that it was possible to quantitate aortic regurgitation by measurements of the distance from the innominate artery that a catheter had to be placed in the aorta in order that a tracer substance could be detected by means of an ear densitometer. He also pointed out that the V wave of the left atrial pressure curve could be used, under proper circumstances, as a measure of mitral regurgitation, particularly when the arterial pressure was elevated by the infusion of norepinephrine. Finally Dr. Braunwald found that in the presence of aortic regurgitation, a competent mitral valve acts in two ways to protect the circulation: (1) it limits the elevation of left atrial and pulmonary capillary pressures and (2) it makes possible the high left ventricular end diastolic pressure as a result of which a more forceful ventricular contraction occurs provided the ventricle is still on the ascending limb of its ventricular function curve.


The panel then considered the coronary blood flow and cardiac energetics as ana-

lyzed by Dr. S. M. Horvath. He pointed out that although no existing method for measuring coronary blood flow in the intact animal is entirely satisfactory, the nitrous oxide method is capable of giving a valuable insight into qualitative changes. He pointed out that appreciable quantities of blood coming from the right coronary appear in the coronary sinus. Dr. Horvath indicated that it was possible to catheterize the right or left coronary arteries by an approach through the aorta and he reported on pressure movements, epicardial electrocardiograms, and the effects of the injection of cold saline. He implied that such observations might have significance in studying disease states. He emphasized the importance of anastomotic channels in the consequences of the occlusion of one or another of the coronary vessels.

The next topic of discussion, the mechanisms of control of the cardiac output in man, was presented by Dr. J. V. Warren. He stressed the fact that in the intact subject with the numerous normal control mechanisms operating there was no regular correlation between ventricular diastolic pressure and cardiac output. He stressed the fact that numerous control mechanisms such as changes in heart rate and alterations in venous return interact to obscure the role of

individual controlling variables. In the discussion it was pointed out that the basic properties of heart muscle and basic capacities of the vascular bed for alterations provide the machinery by means of which numerous nervous and humoral factors can operate to regulate the work of the heart.

On the second day of our conference we had one more presentation when we met again as a single group. This was a discussion by Dr. A. C. Burton, who talked on the importance of the size and shape of the heart. He presented the idea that Laplace's Theorem may have some very important practical implications in the interpretation of cardiac performance. For example, a doubling of the ventricular volume calls for a quadruple increase in the tension of the ventricle per unit area in order to maintain equivalent pressure, when Laplace's Law is applied to the heart. Thus, a dilated heart shows a serious loss of cardiac efficiency because the total cardiac load equals the external work plus the energy cost of maintaining tension in the heart muscle. It is the latter of these which is much the greater. He quoted other workers who have shown that the oxygen requirement of the heart is determined by its diastolic size and is relatively independent of the work it has to do. This, then, he related to Laplace's Law.

ART  CLINICAL PHYSIOLOGY

CHAPTER 11 OCCUPATIONAL AND ENVIRONMENTAL FACTORS IN THE DEVELOPMENT OF HEART DISEASE

Ancel Keys

The general idea that occupation may play a role in the etiology of at least some kinds of heart disease is very old but only recently has it attracted serious attention. As yet the facts available for consideration are few and confusing, the problems and variables of concern are ill defined, and there is no theoretical framework on which hypotheses of pathogenesis can be devised and tested.

If, indeed, occupation is or can be an etiological factor in heart disease, then this is of the utmost importance to discover. For occupations and the manner of life they impose can be altered and a vista of prevention would loom for the future. But the facts of such possible relationships must first be found and the mechanisms involved must be, at least vaguely, appreciated before any useful application can be made. On the other hand, if occupation per se is inconsequential, or is only accidentally associated with other factors of real consequence, then, too, the facts must be discovered so that re-

search may be more profitably concentrated elsewhere.

In any case this Conference has a responsibility to point out the discrepancy between the magnitude and potential importance of the problem and the minute amount of research effort it has received until now. I, at least, am impressed with how little we can now say about the virtues or hazards, in regard to the development of heart disease, of muscular exercise or its lack, of thermal and other physical environmental stresses, of habitual posture at work, of job responsibility and emotional tensions associated with occupation.

Before we proceed further with the discussion of facts now known or alleged connecting occupation with heart disease, it is necessary to distinguish and define categories of heart disease.

Because this discussion is concerned about occupation in regard to etiology, I propose to say nothing about congenital, thyrotoxic,

rheumatic, or syphilitic heart disease. Though different occupations may afford more or less opportunity to acquire rheumatic fever or syphilis, so far as I know there are no relationships of these diseases with occupation per se, independent of general economic and social circumstances. And I find it difficult to imagine any close relationship of occupation to the production of thyrotoxic heart disease in this generation or congenital heart disease in the next.

Cor pulmonale and the heart disease that may result from occupation-induced pulmonary pathology would seem to be appropriate for our discussion. But I shall leave this for others, partly because the development chain of events does not seem to conceal many great mysteries, partly because I have little personal experience in this field.

TABLE 11-1 PERCENTAGES OF ALL HEART DISEASE DEATHS AMONG WHITE MEN AND WOMEN IN U.S.A., 1954

Cause	40-44 years		50-54 years		60-64 years	
	Men	Women	Men	Women	Men	Women
420*	78.7%	41.0%	82.5%	55.7%	80.5%	66.7%
422†	2.8	5.7	3.0	5.9	4.8	6.8
440-443‡	4.6	12.6	5.6	17.7	7.9	16.8
Total	86.1%	59.3%	91.1%	79.3%	93.2%	90.3%

* 420, Coronary heart disease

† 422, Other myocardial degeneration

‡ 440-443, Hypertensive heart disease

Actually, as Table 11-1 shows, all these exclusions still leave us for consideration the causes for disease in the vast majority of heart patients. Among U.S. white males from 40 to 70 years of age, coronary heart disease accounts for over 80 per cent of all heart deaths. If we add "other myocardial degeneration," which presumably involves coronary insufficiency in many instances, and hypertensive heart disease, more than 90 per cent of all heart mortality among men of these ages is covered. Among women rheumatic fever is relatively more important than it is among men, but even so from age

40 through 69 these categories of International List numbers 420, 422, and 440-443 account for about 85 per cent of all heart deaths.

Obviously a major focus must be on coronary heart disease, at least in the United States. Elsewhere I have assembled much evidence indicating that in this disease the diet, and particularly its fat content, is an important etiological factor^{1,2,3}. But if we presume that the characteristics of the diet are not necessarily dependent on the occupation, we must inquire to what extent non-dietary factors associated with occupation may be involved in etiology.

Two major theories have been suggested as to how the occupation may influence the development of coronary heart disease and they are to a certain extent interlocking. It is suggested that physical exercise may be protective or, to put it in another way, that inactivity predisposes to the disease^{4,5,6}. The other theory, which has been critically discussed by Arnott,⁷ is that "tension," responsibility, and emotional stress, such as may be involved in some occupations, promotes the development of both coronary heart disease and hypertension. Since most jobs judged to be highly responsible on the basis of the pecuniary awards attached to them entail a relatively sedentary existence, both theories agree that business and professional men have an occupational hazard in regard to heart disease. Incidentally, this concept implies that economic success carries a health risk and therefore may appeal to some moralists as an illustration of the "law of compensation."

The origin of these concepts about activity is primarily in mortality analyses published some 25 years ago by the Registrar General of England and Wales. Vital statistics in Britain are tabulated according to social class—the higher the social class, the less physical work, and, incidentally, the more money. When coronary heart disease began to occupy an important place in British mortality, the Registrar General called attention to the fact that the reported rates varied according to the social class. So far as I know, no effort was

made to see whether these reported differences were real and not merely reflections of different levels of diagnostic skill and fashion applied to practice with different socioeconomic classes and in different regions. The distribution of social and economic classes in Britain is not geographically uniform.

TABLE 11-2. MORTALITY IN 1930-32 PER 100,000 OF MEN AGED 35-64, ENGLAND AND WALES SOCIAL CLASSES III, IV, V, AS ANALYZED BY MORRIS AND HEADY ACCORDING TO PHYSICAL ACTIVITY OF OCCUPATION

Ascribed cause of death	Heavy work	Intermediate and doubtful	Light work
All causes	2104	2578	2376
Accidents	98	75	50
Respiratory tuberculosis	102	165	141
Bronchitis	101	124	79
Pneumonia	117	166	135
Valvular heart disease	159	175	154
Coronary " "	40	53	87
Myocardial disease	230	284	244

As shown in Table 11-2, Morris and Heady³ reclassified the lower three social classes in Britain according to the physical demands of their occupations and computed mortality rates for 1930-32. Though the mortality rate ascribed to coronary heart disease was twice as high among the light workers as among heavy workers, even among the light workers this was rarely the ascribed cause of death in 1930-32. Myocardial disease, most of which undoubtedly would now be classed as coronary, was far more important. I suggest that in these often-cited old figures there is no real evidence as to what was the incidence in 1930-32 of coronary heart disease nor any good basis to suggest it was related to physical activity.

Classification of all men in a country according to physical activity results in heterogeneity within classes in regard to other factors so that epidemiological conclusions about the effect of activity are hazardous. A less questionable analysis is shown in Table 11-3. Morris and his colleagues studied postal and telegraph employees in London more

TABLE 11-3. INCIDENCE OF CORONARY HEART DISEASE AMONG POSTAL AND TELEGRAPH EMPLOYEES IN LONDON AGE-STANDARDIZED RATES PER 10,000 MEN (MORRIS *et al* ⁴)

Activity	Occlusion	Total incidence
Postmen	6	18
Intermediate	7	20
Sedentary	7	24

recently and found the total incidence of all forms of coronary heart disease to be 25 per cent lower among the mailmen who deliver the mail on foot or on bicycle than among the employees who work seated.⁴ The incidence of occlusions, however, was not significantly related to physical activity.

In a study on London busmen, however, as shown in Table 11-4, more significant dif-

TABLE 11-4. INCIDENCE OF CORONARY HEART DISEASE AMONG EMPLOYEES (BUSMEN) OF THE LONDON TRANSPORT EXECUTIVE AGE-STANDARDIZED ANNUAL RATES PER 10,000 MEN AGED 35-64 (MORRIS *et al* ⁴)

Occupation	Occlusions	Total incidence
Drivers	15	27
Conductors	8	19

ferences were found. The bus drivers, who work seated, had almost twice the frequency of both occlusions and total coronary heart disease of the conductors who work standing and frequently climb the steps of the double-decker buses. The question is whether the difference in coronary incidence may be ascribed to difference in the activity on the job. We should note, incidentally, that there are no data on the energy expenditure of the drivers versus the conductors; the drivers, though seated, are certainly not sedentary in the sense that a clerk is.

A major objection to all such analysis is the implicit assumption that the men classified in regard to activity are alike in other respects. The drivers and conductors represent the same social and economic level but, as Figure 11-1 shows, they appear to differ in at least one respect even at the outset of their respective careers with the Transport

an urban population living at a much lower average level of activity, had a somewhat lower density of coronary patients

The age and sex distribution of acute infarcts in Finnish Karelia is indicated in Table 11-8. The frequency at relatively young ages,

TABLE 11-8 AGE AND SEX DISTRIBUTION OF ACUTE MYOCARDIAL INFARCTION IN JOENSUU CENTRAL HOSPITAL, FINLAND, IN 1954-55 (Pit Ahrenberg)

Ages	Men	Women
30-39	9	1
40-49	46	8
50-59	41	14
60-69	23	6
70 and over	18	6
All ages	137	35

even among women, is impressive. In this series of 172 cases including 35 women, there were four women under the age of 45.

In most of the world the rural and heavy manual worker populations as well as the entire populations of poor countries have a relatively low incidence of coronary heart disease. We also know that the serum cholesterol concentrations in such groups tend to be correspondingly low. One favored explanation has been that physical exercise is protective and it has been assumed that almost all people in poor countries such as Italy and Japan work very hard at manual labor.

Actually, even in the poorest countries a considerable proportion of the population is sedentary or engages only in light work. Italy and Japan are good examples where the proportion of the population who are shopkeepers, clerks, civil servants, and artisans working at a low level of energy expenditure is large and yet the age- and sex-specific frequency of coronary heart disease in middle age for the entire population is only a fraction of that in the United States, perhaps a fourth in the case of Italy, not over a tenth in the case of Japan.

It appears then that if physical activity or its lack is a factor in the etiology of coronary heart disease, it cannot be very

large; it cannot begin to explain the difference between rural Karelia and urban Fukuoka, in Japan, where our researches in 1956 fully confirmed the Japanese statistics as to very low incidence of coronary heart disease.

TABLE 11-9. MEAN SERUM CHOLESTEROL CONCENTRATION, IN MG. PER 100 ML., IN SEDENTARY JAPANESE MEN AGED 40-49, MATCHED IN BODY FATNESS BY SKIN FOLD THICKNESS, AND AVERAGE PERCENTAGE OF DIETARY CALORIES FROM FATS

Place	Percentage fat calories	Cholesterol
Shime, Japan	13	160.6
Honolulu, Hawaii	32	223.4
Los Angeles, California	40	247.9

As Table 11-9 shows, differences in physical activity do not explain the differences in serum cholesterol observed when we compare samples of populations characterized by differences in coronary disease.¹¹ Japanese men in Japan are very rarely attacked, in Los Angeles the Nisei show no significant difference from the local Caucasians; and in Hawaii the local Japanese are intermediate. Even when we compare men of the same age, matched as to body fatness as well as to habitual physical activity, the serum cholesterol differences persist and correspond to the percentage of fats in the diet. The same general relationships were found in Italy, South Africa, and Sweden.

On closer inspection of data from such population samples (e.g., Japan, Italy, Spain, South Africa) we find some tendency for the serum cholesterol concentration to be lower among heavy workers than among the less active men. But, as indicated in Table 11-10, when the diets are studied we also find a corresponding difference in the proportion of calories from fats.¹⁴

In part, the inverse relationship between occupational activity and the percentage of calories from fats observed in many populations is related to income, those doing heavier work receiving the lowest pay so they are unable to afford the more expensive fats.

foods. Also important is the tendency for men to eat relatively constant amounts of meats and fatty foods and to adjust total calories according to need by altering the consumption of carbohydrate staples—bread, potatoes, rice, and the like.

TABLE 11-10 MEAN TOTAL CHOLESTEROL IN MG PER 100 ML. OF SERUM OF JAPANESE MEN AGED 40-49 IN FUKUOKA PREFECTURE, KYUSHU, CLASSIFIED ACCORDING TO OCCUPATION, TOGETHER WITH AVERAGE PERCENTAGE OF DIETARY CALORIES FROM FATS

Occupation	Number of men	Serum cholesterol	Percentage fat calories
Sedentary, doctors	52	196.6	22.0
Sedentary, clerks, teachers	25	159.8	16.1
Moderate to heavy, miners	57	145.6	10.3
Very heavy, peasant farmers	48	141.5	9.7

* Money income of the miners is more than that of the clerks and teachers.

It is possible to speculate ad infinitum that exercise, per se, should reduce the risk of coronary thrombosis or the severity of its effects. The effect of exercise in improving vascularity is often suggested. Perhaps exercise after an occlusion may help to develop collaterals but before occlusion the effect seems to be to reduce the vascularization per unit mass of myocardium,¹³ vascularity does not keep pace with hypertrophy. Theoretically, a better argument for exercise may be developed on the idea that stasis promotes thrombosis and therefore a higher average rate of blood flow, such as produced by exercise, is desirable. But there is no real evidence in regard to coronary heart disease.

The evidence about hypertension is even less satisfactory. Though many suggestions and claims have been made about the etiology of hypertension, the available information does not, at present, warrant any extended discussion as to the possible influence on the incidence of activity, of occupation, or even of the diet apart from total calories. Obesity seems to predispose to hypertension. Study of the extensive literature on the blood pressure in various racial, geographic, and occupational groups merely indicates the

need for more critical large-scale studies and the fact that hypertension is common in all populations.

In Russia an extensive study has been in progress for some years. Professor Mjasnikov's survey of over 200,000 workers is interesting but we must await critical evaluation of the data and methods before coming to any reliable conclusions. In general, the Russians claim the highest frequency of hypertension occurs among executives, telephone operators, and telegraphers, and they attribute this to psychic causes. They report the lowest frequency among employees in oil refining and various metal industries and they suggest that toxic factors may be involved in producing relative hypotension.

CONCLUSIONS

Few conclusions can be drawn at present about the influence of occupation or physical activity on the development of either coronary heart disease or hypertension. Such influences as have been claimed in regard to coronary heart disease are of dubious validity, to say the least, because of possible bias in the samples and the simultaneous operation of other variables such as the diet. The present indications are that physical activity or its lack is not, per se, a major etiological factor.

BIBLIOGRAPHY

1. KEYS, A., ANDERSON, J. T., FIDANZA, F., KEYS, M. H., and SWAHN, B. Effects of diet on blood lipids in man, particularly cholesterol and lipoproteins, *Clin Chem* 1: 34, 1955.
2. KEYS, A., and ANDERSON, J. T. "The Relationship of the Diet to the Development of Atherosclerosis in Man," pp. 181-197 in *Symposium on Atherosclerosis*. Washington, D.C., Nat'l Acad. Sc., Nat'l Res. Council, Publ. 338, 1954.
3. KEYS, A. The diet and the development of coronary heart disease, *J. Chronic Dis.* 4: 364, 1956.
4. STOCKS, P. Coronary disease and modern stress, *Lancet* 1: 351, 1951.

- 5 MORRIS, J. N., and HEADY, J. A.: Mortality in relation to physical activity of work; preliminary note on experience in middle age, *Brit. J Indust. Med* 10 245, 1953
- 6 MORRIS, J. N., HEADY, J. A., RAFFLE, P. A. B., ROBERTS, C. G., and PARKS, J. W.: Coronary heart disease and physical activity of work, *Lancet* 2 1053 and 1111, 1953.
- 7 ARNOTT, W. M. The changing etiology of heart disease, *Brit M J* 2 887, 1954
- 8 MORRIS, J. N., HEADY, J. A., and RAFFLE, P. A. B. Physique of London busmen: epidemiology of uniforms, *Lancet* 2 569, 1956
- 9 DAWBER, T. R., MOORE, F. E., and MANN, G. V. "Coronary Heart Disease in the Framingham Study," in *Symposium Measuring the Risk of Coronary Heart Disease in Adult Population Groups Am J Publ Health* 47 2 (Supp.), 1957
- 10 BJORCK, G., OVERBECK, W., and GRONVALL, C. Coronarkrankheit und Herzinfarkt in Malmo (Ein Beitrag zur geographischen Pathologie der Coronarkrankheit), *Cardiologia* 25 232, 1954
- 11 MORRIS, J. N., HEADY, J. A., and BARTFY, R. G. Coronary heart disease in medical practitioners, *Brit M J* 1 503, 1952
- 12 CHAPMAN, J. M., GOERKE, L. S., DEXON, W., LOVFLAND, D. B., and PHILLIPS, E.: "The Clinical Status of a Population Group in Los Angeles Under Observations for Two to Three Years," in *Symposium: Measuring the Risk of Coronary Heart Disease in Adult Population Groups, Am. J. Publ Health* 47 33 (Supp.), 1957.
- 13 Life Insurance Companies' Inst Med Stat, Oslo City Hosp. Rep No 5. Myocardial infarction: an epidemiologic and prognostic study of patients from five departments of internal medicine in Oslo 1935-1949, *Acta Med. Scandinav. Supp* 315, pp 1-58, 1956 (v.154).
- 14 KEYS, A., ANDERSON, J. T., ARESU, M., BJORCK, G., BROCK, J. F., BRONTE-STEWART, B., FIDANZA, F., KEYS, M. H., MALMROS, H., POPPI, A., POSTELL, T., SWAHN, B., and DEL VECCHIO, A. Physical activity and the diet in populations differing in serum cholesterol, *J Clin Invest* 35-1173, 1956
- 15 HAKKILA, J. Studies on the myocardial capillary concentration in cardiac hypertrophy due to training: an experimental study with guinea pigs, *Ann med. exper & biol Fenniae, Supp.* 10, pp 1-82, (v33).

CHAPTER 12 RELATIONSHIP OF PHYSICAL ACTIVITY TO SERUM CHOLESTEROL CONCENTRATION

Henry Longstreet Taylor

J. N. Morris¹ has shown that certain occupational groups whose job requires moderate physical activity through middle age, i.e., bus conductors and postmen, have a lower mortality from coronary heart disease and are more apt to survive their first coronary closure than sedentary occupational groups such as bus drivers and telephone operators. Furthermore, Keys and his collaborators^{2,3} have shown that in Sweden, South Africa, and other countries the men who hold active jobs and, therefore, expend a larger amount of energy are characterized by lower serum cholesterol concentrations than their sedentary contemporaries. These observations raise the question of whether physical activity per se has an effect on the serum cholesterol concentration and, in this way, may be presumed to influence the rate of atherogenesis.

The acute effects of mild exercise have been studied and it has been demonstrated that grade walking on the motor-driven treadmill prevents the small but significant

rise in serum cholesterol concentration that occurs after a meal containing fat and definitely reduces the increase in serum cholesterol concentration that follows alimentary cholesterol loading.⁴

The study of the chronic effects of increasing the daily energy expenditure by physical activity has been approached by Mann and his associates⁵ who examined the question in three subjects. The daily caloric intake of these men was increased by approximately 3000 calories to the level of 6000 calories a day for one month. The grams of fat in the diet were held constant during both the control and activity periods. This dietary prescription reduced the percentage of calories supplied by fat by approximately half. The subjects in this experiment made heroic efforts to burn up the additional 3000 calories by physical activity. However, they were, apparently, not fully successful since they gained from three to five pounds in weight. From the experience gained in the Labora-

tory of Physiological Hygiene, one would expect that such a gain of weight would result in an increase in the concentration of serum cholesterol provided physical activity per se had no effect. In a study of weight gain from overeating, Anderson, Lawler, and Keys⁶ found that men who gained 6.3 pounds in five weeks sustained an average serum cholesterol rise of 18.6 mg per 100 ml of serum. On the other hand, reducing the percentage of calories supplied by fat without changing the physical activity or the caloric intake resulted in a decline of serum cholesterol concentration of 20 mg. per 100 ml serum.⁷ One might predict, then, that if physical activity had little or no effect on the serum cholesterol concentration, Mann and his co-workers would observe only very small effects as the result of their experimental conditions. This was in fact what they found, i.e., in the three subjects the serum cholesterol concentration either remained unchanged or showed a slight decline during the month of high physical activity at 6000 calories.

With this background, an experiment was designed to test the hypothesis that, other things being equal, the serum cholesterol concentration is controlled by the percentage of total calories derived from fat, and is independent of the absolute fat intake when the total caloric intake is increased by physical activity, body weight being held constant.⁸ The plan was to stabilize nine clinically healthy students on a diet containing 3250 calories per day with enough fat (principally animal fat) to account for 42 per cent of the total calories. At the end of a two-week control period during which the men followed their usual physical activities, blood samples were drawn with the men in the basal state on two successive days. The men spent 10 days becoming accustomed to walking on the motor-driven treadmill. The subjects then walked two hours a day at 3.5 miles per hour on a 10 per cent grade six days a week for 17 days. This work consumed 1280 calories a day. An allowance of 330 calories was made for the basal metabolism and physical activity that would have

been undertaken if the men had not spent the time on the treadmill. The diet was increased 950 calories in such a way that the per cent of total calories derived from fat was not changed but the amount of fat was increased from 155 to 201 grams per day. At the end of this period two blood samples were drawn on successive days. The treadmill walking was terminated and the men returned to the diet of the control period for one week, at the end of which time two more blood samples were drawn.

The mean weight of the group at the start was 171.0 pounds and 170.8 at the end. It did not vary by more than 1.2 pounds during the entire experiment. The mean serum cholesterol concentration was 158.3, 159.8 and 161.5 mg per 100 ml of serum at the end of the control, exercise, and post-exercise sedentary periods, respectively; there was no statistically significant change in the beta lipoprotein cholesterol or the nonesterified fatty acids during the entire experiment. The phospholipids showed a small but statistically significant drop after the exercise period.

Two additional experiments following the same general experimental design have been carried out. In the second experiment, the calories added to pay for the cost of exercise were all in the form of fat. Care was taken to provide a fat that had the same proportion of saturated to unsaturated fatty acids as were present in the food fed during the control period. In the third experiment the exercise calories were paid off with 100 per cent carbohydrate. The results are summarized in Table 12-1. It will be observed that the expected increase in serum cholesterol concentration did not occur in Experiment 2. It is clear from Experiments 1 and 2 that substantial amounts of fat (46 gm in Experiment 1 and 102 gm in Experiment 2) can be added to the diet to maintain caloric balance without raising the serum cholesterol concentration when the level of physical activity is increased. When physical activity is held constant and the composition of the diet is varied, the effects on the serum cholesterol concentration are sufficiently constant to allow the prediction of the change that

TABLE 12-1 THE EFFECT OF INCREASE IN THE LEVEL OF DAILY PHYSICAL ACTIVITY BY 1200 CALORIES ON THE SERUM CHOLESTEROL LEVEL IN THE PRESENCE OF DAILY FOOD SUPPLEMENTS OF 900-950 CALORIES OF DIFFERENT COMPOSITION IN EACH CASE THE CONTROL DIET CONTAINED 3200 CALORIES, 40 PER CENT OF WHICH WAS DERIVED FROM FAT

Experiment number	Per cent calories from fat in food supplement	Per cent calories from fat—total food	Expected change in cholesterol concentration	Observed change in cholesterol concentration
1	40	40	0	0
2	100	53	+28 mgs	0
3	0	31.8	-20 mgs	-18

will occur in the serum cholesterol when the fat content or the type of fat is altered in the diet.⁹ If the physical activity were held constant in Experiment 2 and the percentage of fat from calories was increased from 40 to 50 per cent, we could be confident that the serum cholesterol would increase. So it may be said that the physical activity in Experiment 2 altered the expected response to increasing the proportion of calories derived from fat. This finding underscores the necessity of holding physical activity constant, if the prediction equations proposed by Keys, Anderson, and Grande⁹ are to be valid, but in no way challenges the results reported by these investigators. It does confirm the conclusions of Mann and his associates⁶ that exercise appears to prevent an increase in serum cholesterol. These investigators showed that if exercise is stopped and calories continued, the serum cholesterol increased. This appeared to some to be another way of saying that a positive caloric balance increased the serum cholesterol. The effects of exercise might be looked on as nonspecific but, in the situation reported here, exercise has apparently prevented a rise in serum cholesterol that might be expected on grounds other than that connected with the maintenance of caloric balance. This suggests that exercise may have a specific effect on cholesterol metabolism.

The effects of diet on the level of the serum cholesterol concentration in situations

where the total calories expended by the individual remains constant can be understood, if the lipoprotein cholesterol complex is regarded as a vehicle for fat transport. The role of lipoprotein in fat transport has been proposed by Anfinsen¹⁰ Anderson, Lawler, and Keys⁸ have pointed out that if one regards the route of transport in which lipoproteins play a role as that from gut to liver to fat depot, the effects of diet may be explained by assuming that an overload on the transport system (lipoprotein concentration per unit of circulation) will result in an increased lipoprotein cholesterol complex concentration and an underload will result in a fall of the concentration of lipoprotein cholesterol complex. The results reported here have raised difficult problems of interpretation of current concepts of fat transport, and these have been discussed in detail elsewhere.¹¹ It appears that the lipoprotein cholesterol complex transport mechanism is largely by-passed in exercise. Further work is clearly necessary to clear up some of the contradictions that appear in this area.

Elsewhere, Keys and his associates⁸ have pointed out that men performing heavy industrial work in Sweden, South Africa, and the United States tend to pay off the energy required by physical activity with foodstuffs that are largely carbohydrate, i.e., bread, potatoes, and other staples, such as beans. This results in a lower percentage of total calories accounted for by fats in the diet of heavy workers than in the diet of sedentary individuals in the same area. Experiment 3 supports the suggestion that in this situation the serum cholesterol may be lower in workers in heavy industry than among clerical employees. Higher serum cholesterol in sedentary workers than in heavy industrial laborers have actually been observed in Sweden² and South Africa.³ However, a physically active job may not always result in a low serum cholesterol and this is illustrated by some preliminary results that we have recently obtained on railroad clerks and switchmen. Here there appears to be little or no difference in serum cholesterol concentration between the physically active switchmen

and the sedentary clerks. Forty-seven clerks between the ages of 40 and 49 years had a mean serum cholesterol concentration of 229.6 ± 6.7 mg per 100 ml. of serum, and 51 switchmen showed 228.7 ± 5.7 mg. per 100 ml of serum.

We have been concerned here with the effect of exercise in the control of the serum cholesterol concentration. This is of concern because it is widely believed that atherogenesis proceeds more rapidly in the presence of a high serum cholesterol concentration as compared to a low concentration. The effects of exercise on the serum cholesterol concentration do not appear to be large enough, in themselves, to account for the results observed by J. N. Morris in England. Differences in dietary composition of the two groups could be an important factor. The work of Blumgart and his collaborators¹² and of Eckstein¹³ on the existence of coronary artery anastomoses and the role of exercise in promoting them provides us with clues as to the possible utility of exercise as a preventive factor in the etiology of coronary heart disease. However, as Keys¹⁴ has pointed out elsewhere, it is necessary to know something of the physiological characteristics of active and sedentary populations, which are known to differ in the prevalence of coronary disease, before we can begin to propose sound theories as to the mechanisms producing the advantages, if any, that physical activity bestows on the potential victim of coronary disease.

THE CONSTRUCTION AND USE OF TESTS OF PHYSICAL FITNESS

Physical fitness tests that are being used currently to assess the response of the cardiovascular system to the stress of physical work all suffer from one or more of the following faults:

1. Skill in performing the work task around which the test is constructed makes up a large fraction of the differences between individuals;
2. The rate of work is too low so that measurements of pulse or respiratory func-

tion are not reliable indexes of the response to work;¹⁵

3. The measurements, usually pulse and a function of respiration, are affected by emotion and the effects of emotion cannot be distinguished from a poor cardiovascular or respiratory response to physical work;¹⁶

4. Pulse rates are counted during recovery instead of during actual work. Recovery pulse rates are valid measures of a change in state of a given individual from one time to another but a poor measure of the difference between individuals;¹⁷

5. An important dimension of the actual measurement is the length of time work can be sustained. This is a self-determined end point and is subject to a great variety of factors including emotion, attitudes toward work, and the relationship between the investigator and the subject;¹⁸

6. "Physical Fitness Indices" are used that combine a number of measurements and give equal weight to each measurement without proper validation of the weights assigned;¹⁹

7. Tests that are relatively free from the above faults require maximum co-operation from the subject and are too strenuous to apply to most individuals suffering from cardiac disease.^{19,20}

It is generally agreed that skill in treadmill grade walking is highly developed in males and that differences in mechanical efficiency are very small. A similar statement appears to be correct for the bicycle ergometer in those countries such as Denmark and Holland where bicycles are widely used as a means of transportation.

In normal young men there is much evidence to indicate that if one is going to get reasonable differentiation between individuals who are in good physical condition and those who are not by measuring the pulse rate response to a fixed task, one must use a rate of work which is roughly half of the maximal oxygen intake.²¹ This rate of work usually elicits a pulse rate of from 120 to 150 beats per minute. Since cardiac patients show a very wide range of maximal oxygen intake and work capacity, it would seem that the investigator of fitness of car-

diacs should be prepared either to examine several rates of work or to measure the amount of work necessary to raise the pulse to some predetermined level. Balke²² has been employing such a procedure for the determination of fitness among air force officers. In this procedure, the subject walks at 3.5 miles per hour on the motor-driven treadmill. Pulse rate, blood pressure, ventilation, and oxygen consumption are measured every minute. At the end of each minute the treadmill grade is increased by 1 per cent. In the clinically healthy individual, this procedure is continued until the pulse reaches 180 beats per minute. The discomfort accompanying excessive rates of ventilation may become a limiting factor.²³ Adapting this procedure to the study of cardiac patients would have the advantages of utilizing a task at which most subjects have developed a high degree of skill, the final level of work would be determined by the response of the patient's cardiovascular or respiratory system and it would eliminate the undesirable self-determined end point. Those patients who suffer from a definite lack of motivation would be identified since they would fail to push themselves to the selected end point. The procedure would probably be subject to errors produced by acceleration of the pulse by emotion. Using this or any other submaximal test as a device to predict performance on a job on which the general level of oxygen consumption is known will always be difficult since the skill factor²⁴ on most real life jobs is large and the static work component¹⁶ on most jobs in this country is largely unknown.

In specialized situations, certain tests that cannot be applied to all patients may have useful application. An example is the maximal oxygen intake^{19, 20} procedure which, although it measures the capacity of the cardiovascular respiratory system to move oxygen to the muscles and is free of many of the faults of other fitness tests, is too strenuous to be applied to most patients in its present form. However, Mitchell and Chapman²⁴ have reduced the rate of work and successfully applied this procedure to the

study of patients with valvular disease. They have found that this is particularly useful in assessing the effects of surgery designed to repair valvular defects.

BIBLIOGRAPHY

- MORRIS, J. N., HEADY, J. A., RAFFLE, P. A. B., ROBERTS, C. G., and PARKS, J. W. Coronary heart disease and physical activity of work, *Lancet* 2 1053 and 1111, 1953.
- KEYS, A., ANDERSON, J. T., FIDANZA, F., KEYS, M. H., and SWAHN, B. Effects of diet on blood lipids in man, *Clin. Chem.* 1 34, 1955.
- KEYS, A., ANDERSON, J. T., ARESU, M., BJORCK, G., BROCK, J. F., BRONTE-STEWART, B., FIDANZA, F., KEYS, M. H., MALMROS, H., POPPL, A., POSTELL, T., SWAHN, B., DEL VECCHIO, A. Physical activity and the diet in populations differing in serum cholesterol, *J. Clin. Invest.* 35 1173, 1956.
- KEYS, A., ANDERSON, J. T., and MICKELSEN, O. Serum cholesterol in men in basal and non-basal states, *Science* 123 29, 1956.
- MANN, G. V., TEEL, K., HAYES, O., McNALLY, A., and BRUNO, D. Exercise in the disposition of dietary calories, *New England J. Med.* 253 349, 1955.
- ANDERSON, J. T., LAWLER, A., and KEYS, A. Weight gain from overeating, serum lipids and blood volume, *J. Clin. Invest.* 36 81, 1957.
- ANDERSON, J. T., and KEYS, A. Dietary fat and serum cholesterol, *Fed. Proc.* 12 169, 1953.
- TAYLOR, H. L., ANDERSON, J. T., and KEYS, A. Physical activity, serum cholesterol and other lipids in man, *Proc. Soc. Exper. Biol. & Med.* 95 383, 1957.
- KEYS, A., ANDERSON, J. T., and GRANDE, F. Prediction of serum cholesterol responses of man to changes of fats in the diet, *Lancet* 2 959, 1957.
- ANFINSEN, C. B., JR. Lipoprotein metabolism in the etiology of atherosclerosis, *Minnesota Med.* 38 767, 1955.
- TAYLOR, H. L. Diet, physical activity and the serum cholesterol concentration, *Minnesota Med.* 41.149, 1958.
- BLUMGART, H. L., SCHLESINGER, M. J., and DAVIS, D. Studies on relation of clinical manifestations of angina pectoris, coronary thrombosis, and myocardial infarction to

- pathologic findings, with particular reference to significance of collateral circulation, *Am Heart J* 19:1, 1940
13. ECKSTEIN, R. W. Effect of exercise and coronary arterial narrowing on growth of interarterial coronary anastomoses, *Fed Proc.* 15:54, 1956
 14. KEYS, A. Occupational and environmental factors in the development of heart disease, *First Wisconsin Conference on Work and the Heart*, 1957.
 15. SCHNEIDER, E. C., and TRUESDELL, D. Effects on circulation and respiration of an increase in carbon dioxide content of blood in man, *Am J Physiol* 63:155, 1922.
 16. TAYLOR, H. L., and BROZEK, J. *Unpublished observations*
 17. RHYMING, I. Modified Harvard step test for evaluation of physical fitness, *Arbeitsphysiologie* 15:235, 1953
 18. JOHNSON, R. E., BROUHA, L., and DARLING, R. C. Test of physical fitness for strenuous exertion, *Rev can de biol.* 1:491, 1942
 19. TAYLOR, H. L., BUSKIRK, E., and HENSCHEL, A. Maximal oxygen intake as an objective measure of cardio-respiratory performance, *J. Appl. Physiol* 8:73, 1955
 20. AASTRAND, P.-O.: *Experimental Studies of Physical Working Capacity in Relation to Sex and Age* Copenhagen, Munksgaard, 1952.
 21. AASTRAND, P.-O. Human physical fitness with special reference to age and sex, *Physiol Rev.* 36:307, 1956.
 22. BALKE, B.: Optimale körperliche Leistungsfähigkeit, ihre Messung und Veränderung infolge Arbeitsermüdung *Arbeitsphysiologie* 15:311, 1954
 23. SCHILLING, J. A., HARVEY, R. B., BECKER, E. L., VELASQUEZ, T., WELLS, G., and BALKE, B.: Work performance at altitude after adaptation in man and dog, *J. Appl Physiol* 8:381, 1956
 24. MITCHELL, J. and CHAPMAN, C. B.: Relation of maximal oxygen uptake to cardiac capacity and arterial oxygen saturation in normal subjects and in patients with mitral stenosis, *J Clin. Invest* 36:915, 1957.

CHAPTER 13 HEART DISEASE IN INDIANA FARMERS*

W. H. M. Morris

Over 21 per cent of the white male fatalities certified due to heart disease in Indiana occur in farmers and farm workers, a group that comprises 14.4 per cent of the employed population. Studies by the Statistical Re-

the fatalities in this group do not exceed those expected for the general population.

Similar results were obtained in a study of the fatalities in the year 1952. The conclusion reached was reported as follows: "The

TABLE 13-1 ACTUAL DEATHS FROM HEART DISEASE AS A PERCENTAGE OF EXPECTED DEATHS* FOR INDIANA FARMERS AND FARM LABORERS, 1950
(Statistical Service, Indiana State Board of Health)

	Age group					
	14-34	35-44	45-54	55-59	60-64	65 and over
Percentage of expected fatalities	38.9	41.0	56.4	83.4	86.6	112.6

*Fatalities expected using the age specific mortality rate for all males applied to the population of farmers and farm operators.

search Section of the Indiana State Board of Health¹ made on the fatalities occurring in the year 1950 illustrate this but show that

*The studies described herein are part of the Purdue Farm Cardiac Project, a co-operative study, which is in part supported by the Indiana Heart Foundation and the American Heart Association. Journal Paper No. 1169 of the Purdue Agricultural Experiment Station.

appears extremely unlikely that the death rate is independent of occupational grouping. Figures such as obtained are the result by chance much less than one in a thousand. It should be stated, however, that it is possible that other factors such as actual variations in the occupational death rate could be responsible for the

occurrence." Other studies of the effect of occupational factors upon the development of heart disease have been critically reviewed by other participants in the First Wisconsin Conference on Work and the Heart.

AGE DISTRIBUTION IN THE FARM POPULATION

The 1950 Census of Population² reported that 14.39 per cent of the white male labor force was occupied in farming. However,

were made available to farmers; this will lead to an earlier retirement among farm operators in the near future. Since the first farmers retired under the scheme in July, 1956, the impact on retirement is still uncertain.

AGE AND CARDIAC FATALITY

Deaths from heart disease occur at a later age in the farm than in the nonfarm males. The results of a study of five years of fatal-

TABLE 13-2 EMPLOYMENT IN FARMING AS A PERCENTAGE OF ALL EMPLOYED WHITE MALES, INDIANA, 1950

Per cent employed	Age group									
	Under 25	25-29	30-34	35-44	45-54	55-59	60-64	65 and over	All ages	
Farm	14 14	9 39	10 44	11 37	14 28	18 18	21 28	30 52	14 39	
Nonfarm	85 86	90 61	89 56	88 63	85 72	81 82	78 72	69 48	85 61	
Total	100 00	100 00	100 00	100 00	100 00	100 00	100 00	100 00	100 00	

TABLE 13-3 INCIDENCE OF CARDIAC FATALITIES BY CAUSE IN FARM AND NONFARM WHITE MALES, INDIANA, 1949-1953

Primary cause groups		Fatalities per year				Median age of fatalities	
Numbers	Description	Nonfarm	Farm	Total	Farm as percentage of total	Non-farm	Farm
410-416	Chronic rheumatic heart disease	263	58	321	18 1	56 93	74 59
420	Atherosclerotic (coronary)	4878	1147	6025	19 1	67 05	74 30
421	Chronic endocarditis (nonrheumatic)	115	39	154	25 4	—	—
422	Other myocardial degeneration	782	296	1078	27 5	73 09	76 28
430-434	Other diseases of the heart	278	88	366	24 0	68 74	77 08
440-443	Hypertensive heart disease	943	314	1257	24 0	72 25	83 67
410-443	All cardiac	7259	1942	9201	21 2	68 07	76 23

30.52 per cent of the employed males of 65 years and older was employed in farming (Table 13-2). The mean age of farm operators has changed little from 50 years in the last 25 years; in 1940 it was 50.2, in 1945, 50.3, in 1950, 49.6, and in 1954, it was 50.5 years.

The work of farm operator or manager permits a man to reduce the scale of operation and to carry on activity to an old age. However, in 1954, Social Security benefits

(over 45,000 individual cases) show the median age at death of the farmer to be eight and a quarter years greater than that of the nonfarmer (Table 13-3). A study of the fatalities in 1953, 1954, and 1955 shows that the farm fatalities occur at a greater age than those among the professional group, technical proprietors, managers, clerical and sales persons, or among the craftsmen, operatives, service workers, and laborers.

The occupational classes are those from

TABLE 13-4A DISTRIBUTION OF FATALITIES FROM ARTERIOSCLEROTIC HEART DISEASE BY AGE, INDIANA WHITE MALES, 1949-1953

Occupational class	Age at fatality								
	Under 25	25-34	35-44	45-54	55-64	65-74	75-84	85 and over	All ages
	Percentage of fatalities								
Farm	02	02	1 19	4 81	16 10	29 94	35 56	12 14	100
Nonfarm	07	64	4 54	14 45	25 28	29 07	19 96	5 03	100

TABLE 13-4B DISTRIBUTION OF FATALITIES FROM ARTERIOSCLEROTIC HEART DISEASE BY AGE FOR THREE OCCUPATIONAL GROUPS, 1953-1955

Occupation group	Year	Age at fatality							
		Under 35	35-44	45-54	55-64	65-74	75-84	85 and over	All ages
		Percentage of fatalities							
Farm	1953	34	1 36	4 59	14 29	30 78	34 61	14 03	100
	1954	15	1 77	3 70	14 80	31 07	34 08	14 42	100
	1955	00	92	4 59	12 46	27 98	39 37	14 68	100
Professional Clerical	1953	64	5 59	13 64	24 19	31 63	19 31	4 91	100
	1954	57	4 36	14 05	25 31	30 93	19 98	4 75	100
	1955	72	3 92	14 07	24 53	30 96	20 30	5 50	100
Craftsmen Operatives Laborers	1953	14	3 63	14 24	28 27	27 10	20 40	6 23	100
	1954	39	5 18	15 54	24 13	30 36	19 41	4 98	100
	1955	38	3 45	13 30	26 93	29 44	19 46	7 03	100

the Census of Population³ and they are grouped as follows:

1. Farmers and farm managers
Farm laborers and foreman
2. Professional and technical workers
Managers, officials, and proprietors (except farm)
Clerical, sales, and kindred workers
3. Craftsmen, foremen, and kindred workers
Operatives and kindred workers
Service, private household, and protective workers
Laborers, except farm and mine

These figures (Tables 13-4A and 13-4B), of course, reflect the age structure of the occupation group. The computation of the incidence rates is possible with some confidence for fatalities in the age groups below 65 years. Over 65 years of age the statistics on the number of people who would be called farmers or farm workers, if they were

to die, are unreliable. Owing to migration into and out of agriculture the *Cohort* system cannot be used without many assumptions.

The incidence rates for those below 65 years (Table 13-5) show consistently lower

TABLE 13-5 INCIDENCE RATES OF WHITE MALE FATALITIES FROM ARTERIOSCLEROTIC HEART DISEASE IN CERTAIN OCCUPATION GROUPS, INDIANA, 1953-1955

Occupation group	Year	Age Group				Total fatalities
		Under 35	35-44	45-54	55-64	
		Fatalities per 1,000 population				
Farm	1953	0 08	0 57	1 87	5 71	
	1954	0 04	0 81	1 67	6 53	
	1955	0 00	0 42	2 08	5 54	
	Av	0 04	0 60	1 87	5 93	732
Professional Clerical	1953	0 02	0 72	3 33	9 78	
	1954	0 05	1 09	4 06	9 35	
	1955	0 05	0 76	3 64	10 88	
	Av	0 04	0 86	3 68	10 00	2027
Craftsmen Operatives Laborers	1953	0 07	1 12	3 31	8 05	
	1954	0 07	0 91	3 58	8 92	
	1955	0 08	0 91	3 96	9 55	
	Av	0 07	0 93	3 62	8 84	3531

rates for the farm group. There appears to be no consistent difference between the two nonfarm groups, the one engaged in physical work and the other typically engaged in mental work. The nonfarm groups are less homogeneous in their physical activity. Some error is introduced by the group of the population who have left the labor force, which is 2.8 per cent in the 35-44-year group, 5.2 per cent in the 45-54-year group, and 14.0 per cent in the 55-64-year age group. This is small under 55 years and unlikely to affect the general trends in the group 55-64-years. The effect would be to increase the population at risk and so decrease the incidence rate.

PREVALENCE STUDIES

The prevalence of heart disease among low income farmers has been estimated from the medical examinations associated with a federal lending agency.³ An over-all rate of 9.5

TABLE 13-7. CLASSIFICATION OF RESPONDENTS TO MAIL SURVEY, CENTRAL INDIANA, 1957

Classification	County number			
	2	3	4	5
	Survey population			
	1450	1443	1335	1281
	Per cent			
Nonrespondents	23.9	20.8	15.9	23.4
Farmers	55.9	65.8	70.7	57.6
Retired farmers	6.8	7.1	4.2	7.1
Others	13.4	6.3	9.2	11.7
Total	100.0	100.0	100.0	100.0
Reporting medically diagnosed heart disease	Per cent of farmer respondents			
Active farmers	11.5	10.8	11.4	11.4
Retired farmers	25.0	25.0	22.8	23.9

per cent had heart disease which had been diagnosed medically (Table 13-7). Of the retired farmers, 24.4 per cent reported heart disease previously diagnosed medically. The average

TABLE 13-6. PREVALENCE OF HEART DISEASE IN LOW INCOME FARM FAMILIES, FARM SECURITY ADMINISTRATION, 1940, ISOLATED AREAS IN THE UNITED STATES

	Age groups									
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65 and over	All ages
Prevalence, percentage	8.7	6.5	11.3	7.5	11.0	12.9	21.3	22.1	23.6	9.5
Number in sample	150	154	177	186	155	163	89	77	72	1223

per cent was reported. The prevalence is shown to be over 20 per cent in the 55-64-year age groups (Table 13-6). The criteria for diagnoses are not fully described.

As a means of obtaining a stratified random sample for survey in the Purdue Farm Cardiac Project, a questionnaire was mailed to farmers in five counties in Central Indiana. The questionnaire and sampling procedure were revised after covering the first county, which is omitted from this presentation. The over-all response to three waves of mailing to 5,867 people was 78.8 per cent. Of the 5,867 people, 62.3 per cent were active farmers, 6.4 per cent were retired farmers, and 10.2 per cent were in other occupations or had left the area. Of the active farmers who responded to the survey, 11.2 per cent reported that they

age of the retired farmers would, of course, be greater than that of the active farmers.

Tabulating the response of the active farmers in three age groups (Table 13-8) shows an arithmetic increase in the proportion reporting heart disease diagnosed previously. However, the mortality rate increases

TABLE 13-8. AGE OF FARMERS REPORTING MEDICALLY DIAGNOSED HEART DISEASE, MAIL SURVEY, CENTRAL INDIANA, 1957

Age	County			
	2	3	4	5
	Percentage reporting			
Under 45	6.8	7.4	7.0	8.2
45-64	11.8	11.2	14.1	13.6
65 and over	18.9	18.3	16.8	20.0

exponentially with age (Table 13-5). It is probable that many of the farmers in the older age groups with diagnosed heart disease go into retirement, which may account for the relatively small increase in the reported prevalence with age. These figures will be revised in the light of a further review of the medical records, using specifically defined criteria

Medical examination of a sample of *farmers 65 years old or less* drawn at random* from the respondents to the mail questionnaire showed that 44.1 per cent of those reporting heart disease in fact had heart disease,† and a further 10.9 per cent possibly had heart disease. This can be extrapolated to the population of farmers that responded to the mail survey to give a prevalence rate shown in Table 13-9

TABLE 13-9 PRELIMINARY ESTIMATE OF THE PREVALENCE OF HEART DISEASE AMONG FARMERS IN FOUR COUNTIES IN INDIANA; FROM SURVEY AND MEDICAL EXAMINATION

	With diagnosed heart disease	With doubtful diagnosis of heart disease
	Percentage of farmers responding	
Reported diagnosed cardinals*	45	11
Reported not diagnosed cardinals	42	30
Total	87	41

* Reported in medically diagnosed heart disease in response to mail survey

SUMMARY

The apparently high incidence of cardiac fatalities among Indiana farmers is attributed

* There was a geographical bias in the sampling technique, therefore, in the strict sense, the sample was not drawn at random. The extent of the bias is thought to be slight.

† That is, heart disease within the criteria used in the United States Public Health Service long-term epidemiological study at Framingham, Mass.

to the high average age of the group compared to the rest of the labor force. Of all employed, the farmer group comprised 14.4 per cent, but of those employed when over the age of 65 years, 30.5 per cent were farmers.

The median age at fatality of the farmers was eight and a quarter years greater than that of the nonfarmers.

The proportion of cardiac fatalities among farmers occurring in each age group lagged one 10-year age group behind that of nonfarmers.

The incidence rate of fatalities in the farm group also lagged one 10-year age group behind the professional and sales occupation group and the manual and service worker group.

In response to a mail questionnaire, 11.2 per cent of the farmers reported heart disease that had been diagnosed medically. Preliminary analysis of the medical examination of a sample of farmers under 66 years old drawn from this group showed that 44 per cent of those reporting heart disease actually had heart disease and a further 11 per cent may have had heart disease. The prevalence of undiagnosed heart disease in the same sample appeared to be about equal to that of diagnosed heart disease.

BIBLIOGRAPHY

- 1 CAIHOUN, R. A., and ANDERSON, W. *Actual and Expected Deaths from Heart Diseases by Occupation and Ages*, 1950. Indiana St. Bd. Health, Statistical Service, 1953.
- 2 U.S. CENSUS BUREAU, *Census of Population* 1950, Bulletin P-C14, table 76. U.S. Gov't Printing Office, 1952.
- 3 GROVER, M. Physical impairment of members of low-income farm families: 11,490 persons in 2,477 rural families examined by the Farm Security Administration, 1940. Variation of blood pressure and heart disease with age, and the correlation of blood pressure with height and weight, *U.S. Publ. Health Rep.* 63:1083, 1948.

CHAPTER 14 ENERGY EXPENDITURE BY CARDIAC AND NONCARDIAC FACTORY WORKERS*

Herman K. Hellerstein and Amasa B. Ford

INTRODUCTION

A new understanding of the benign outlook for many people with heart disease, particularly for those with coronary arteriosclerosis, has led physicians to permit and even to encourage physical activity that was once thought dangerous.¹ For this new liberality to be applied wisely, however, sound measurement of the stress imposed by various activities is needed.

In an industrial city, the man with heart disease often asks whether to continue, re-

sume, or change his job. In answering this question, the physician may consider not only clinical and social information, but also the physiological response to a standard exertion such as the Master Two-step Test. We have studied this test and found that it requires an energy expenditure of 8.5 Calories per minute for one and a half minutes.² Both rate and duration of energy expenditure are out of proportion to those of ordinary working activity. Even with more prolonged laboratory exercise, as on a treadmill,³ it is impossible to relate the simulated work situation to actual working conditions without specific information concerning the demands of the patient's job. Many measurements of the energy cost of individual activities and a few studies of energy expenditure during several hours or days have been collected by Passmore and Durnin.⁴ However, these studies apply to normal people in other

* This study was supported by a grant from the Cleveland Area Heart Society. The authors express their gratitude to Richard Mackey, Herbert Medoff, James L. Phillips, Frank Tiberio, and Norman Zucker for invaluable assistance in collecting and preparing the data and to the volunteer subjects and personnel of The Cleveland Graphite Bronze Company Division of Clevite Corporation, Jack and Heintz, Inc., and Thompson Products, Inc.

countries and economic conditions. No studies have been published of energy expenditure on the job of patients with heart disease. The present study, therefore, was designed to measure the energy expenditure of factory workers with heart disease, to observe various physiologic parameters of cardiovascular function under actual working conditions, and to compare the workers with heart disease with their healthy co-workers in each particular.

Subjects

A total of 62 subjects was studied on the job. Thirty-six had recognized heart disease, and 26 were normal control subjects. A control subject was matched to each cardiac whenever a healthy worker of comparable age and skill could be found performing the same job in the same plant.

The subjects with heart disease were working employees of three large Cleveland manufacturing firms and were selected through having come to the attention of the industrial physician in the course of employment physical examinations, illness at work, or reports from private physicians following illness. The diagnosis of heart disease was confirmed by a complete medical history, physical examination, and standard twelve-lead electrocardiogram. Twenty-four of the 36 patients with heart disease were evaluated at the Work Classification Clinic of the Cleveland Area Heart Society, where additional laboratory studies including fluoroscopic examination, ballistocardiogram, and the two-step exercise test were performed.

All the subjects were men except for one woman, who had arteriosclerotic heart disease. Among the cardiacs there were 25 with arteriosclerotic heart disease, of whom 20 had old myocardial infarcts confirmed by electrocardiogram. Four subjects had hypertensive cardiovascular disease, five rheumatic heart disease, one luetic aortitis and aneurysm, and one possible congenital heart disease. It is probable that most of the employees with symptomatic coronary artery disease in these three industries were studied. Undoubtedly many individuals with varying

degrees of hypertension and some with asymptomatic rheumatic heart disease were not included. Twelve of the cardiac subjects fell into Class I of the New York Heart Association,⁵ 19 into Class II, and 5 into Class III. Ten of the 36 patients were taking digitalis at the time of the study. The average age of the cardiac subjects was 50.6 years, with a range of 24 to 70 years, while the control group averaged 41.5 years, ranging from 18 to 65 years.

The subjects were found to be working in every major department of the three companies. These plants manufacture many types of light and medium weight metal products, including electric motors and switches, automobile and airplane parts, bearings, and nonferrous metal products. The plants employ a total of 8,500 people and are representative of the type of industry that employed 33.3 per cent of the working population of Cuyahoga County (Cleveland) in a 1946 survey.⁶ Specific jobs studied ranged in skill from janitor to shift foreman and electronics tester, and in energy expenditure from planning clerk to stationary engineer and stock picker (warehouseman). The types of jobs studied are listed in Table 14-1.

TABLE 14-1 SUBJECTS WITH HEART DISEASE
CLASSIFIED BY TYPES OF JOBS

Miscellaneous, 4 subjects	Machine Operators, 7 subjects
Boxmaker	Drill press operator 2 subjects
Laundry crib supervisor	Seaming machine operator
Planning clerk	Set-up burrishing press
Tool crib attendant	Sub lathe operator
Brick workers, 8 subjects	Tool grinder
Assembler	Turret lathe operator
Clutch tester	Maintenance workers, 7 subjects
Electronics tester	Janitor
Gauge setter	Maintenance electrician
Light assembler, 2 subjects	Macaron
Milwright	Painter
Stamper	Plant guard
Farmen, 7 subjects	Stationary engineer
Foreman	Welder
Inspector	Warehousemen 3 subjects
Lead man (foreman)	Stock picker
Maintenance supervisor	
Shift foreman	
Supervisor, 2 subjects	

Methods

Each subject reported to the company dispensary 15 minutes before the beginning

of the shift on a typical working day. The purpose and methods of the study had been explained to each subject at least a day previously. Height and weight were measured, and four copper electrodes were taped in place on the chest in the V_4 and V_6 positions on right and left sides. Lead wires passed through the shirt collar to a socket attached to the subject's belt, where they could be connected to a Sanborn direct-writing electrocardiograph when desired. A pedometer was adjusted and fastened to the subject's belt. An Air Force half-face oxygen demand mask was carefully fitted. The mask was arranged to permit inhalation of room air. From the mask, expired air passed through a plexiglass valve and rubber tubing to the Kofranyi-Michaelis respiratory meter.⁷ The meter, which together with mask and tubing weighs 3.6 kg, was either worn like a knapsack or placed on a table during sedentary observations. While the subject became accustomed to the mask, observations were made of the blood pressure and electrocardiogram. A 5-minute aliquot of expired air was then collected, and pulmonary ventilation was read from the meter in order to determine resting oxygen consumption. Respiratory rate was measured during the collection period. The mask and meter were then removed, leaving the electrodes in place, and the subject was accompanied to his place of work.

A detailed log was kept of the exact duration of each activity in which the subject engaged during the day. Five-minute samples of expired air were collected for the purpose of measuring energy expenditure during each activity, after at least 5 minutes of performing that activity, while wearing the mask and meter. Electrocardiograms and respiratory rate were recorded as the sample was being taken, and the blood pressure was measured either during or immediately after the determination. At least three observations were made of energy expenditure at rest (before work, in mid-shift, and just before the end of the shift). Multiple observations were made on any activity that occupied more than an hour of

the subject's time. Five to nine (average seven) samples of expired air were collected during the day. The specific requirements of the study took about an hour from the regular working time, but in no case did a worker fail to meet a quota for the day or show other objective evidence of hampered efficiency or productivity on account of the study. Additional observations were recorded: of the individual in terms of personality, working methods, and motivation, and of the job in terms of skill, responsibility, adverse working conditions, apparent effort required, and pay.

Samples of expired air were transferred immediately from the butyl rubber collecting bag to glass sample tubes, where they were stored under positive pressure of mercury and thus transported to the laboratory. Analyses for carbon dioxide and oxygen content were performed with the Scholander micro gas analyzer.⁸ Duplicate determinations gave a mean difference of 0.03 volumes per cent (S.D. [standard deviation] of the differences = 0.06 volumes per cent) for carbon dioxide and 0.03 volumes per cent (S.D. of the differences = 0.07 volumes per cent) for oxygen. The volume measurements of the respiratory meter, compared with a 100-liter Tissot spirometer at rates of flow of from 10 to 50 liters per minute, gave a mean error of 1.3 per cent of the total. The maximum resistance to breathing offered by the meter at these flows was 2.0 cm. of water. Oxygen consumption was taken as the product of ventilation and oxygen utilization (percentage of oxygen in room air minus percentage of oxygen in expired air), and converted to energy expenditure (in kilocalories per minute) according to the data of Cathart and Cuthbertson.⁹ All volumes were reduced to standard conditions (0° C, 760 mm Hg, dry). Rates of energy expenditure were expressed without deducting a basal or resting value. Energy expenditure was not divided by body surface area, since these quantities have not been shown to bear a constant relationship during different types of activity.^{10,11}

OBSERVATIONS

Energy Expenditure

The average rate of energy expenditure of workers with heart disease was 1.97 Calories per minute (S.D. 0.48 Calories per minute), or a total of 997 Calories (S.D. 245 Calories) during the 8½-hour work shift. A higher rate of energy expenditure, 2.29 Calories per minute (S.D. 0.64 Calories per minute), obtained during actual working time. The figures for the control subjects were higher but were not significantly different as judged by the *t*-test,¹² using a probability level of 5 per cent ($t = 1.06$, $p = < .4$). The data are summarized and compared with resting values in Table 14-2.

TABLE 14-2. AVERAGE RATES OF ENERGY EXPENDITURE (Calories per minute)

	36 Cardiacs		26 Controls	
	Average	S.D.	Average	S.D.
At rest	1.30	0.25	1.40	0.20
During shift	1.97	0.48	2.13	0.45
During actual work	2.29	0.64	2.51	0.68

The energy cost of individual jobs increased as the work involved the use of progressively larger muscle groups (Fig. 14-1). There was a progression from sedentary to ambulatory, and from handwork to work involving leg, trunk, and shoulder muscles. The same pattern was evident among the control workers. In both groups there was considerable overlap between categories of work. The energy cost of this factory work was remarkably low. Only in the maintenance and warehouseman groups were individuals found whose average rate of energy expenditure equaled twice the resting level.

Maximum rates of energy expenditure varied from individual to individual but were seldom excessive (Fig. 14-2). The noncardiac workers had slightly higher maximum energy expenditures (average peak of 3.45 Calories per minute) than did those with heart disease (average peak of 3.04 Calories per minute). However, when each subject was compared with his matched control, the differences were not statistically significant ($t = 1.56$, $p = < 2$). Different

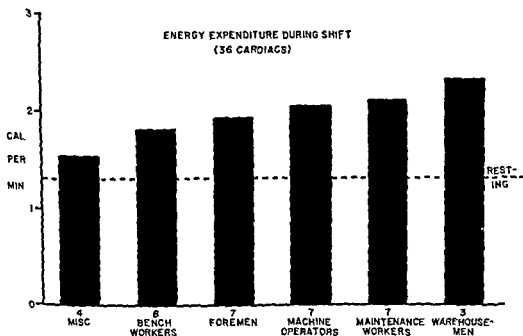


Fig. 14-1 The energy cost of individual jobs in 36 cardiac employees. Note the increasing energy expenditure as progressively larger muscle groups are used.

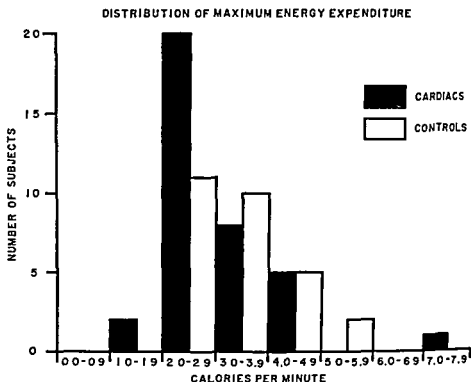


FIG 14-2 Maximum rates of energy expenditure in 36 cardiac workers and 26 noncardiac controls. Although the maximum energy expenditures were slightly higher in the noncardiac employees, the differences were not statistically significant when each subject was compared with his matched control.

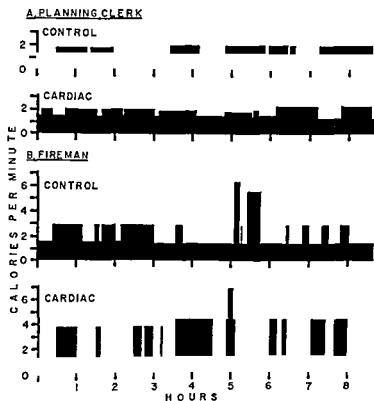


FIG. 14-3 Two basic patterns of energy expenditure. *A* Low rate maintained fairly steadily. *B* High peaks alternating with inactive periods. The frequency and duration of rest periods taken by the cardiac workers and the controls was not significantly different.

jobs required different patterns of energy expenditure. In each job, the magnitude and duration of energy expenditure and sequence of tasks were consistent and were not modified by the presence of heart disease in the worker. Two basic patterns were observed: (1) a low rate of energy expenditure maintained fairly steadily, and (2) high peaks of energy expenditure alternating with inactive periods. Examples of the two patterns are shown in Figure 14-3. The majority of jobs studied resembled the low steady pattern. Only 13 subjects had peaks of energy expenditure above 40 Calories per minute. The workers took an average of nine to ten breaks during the shift, for an average total of an hour and three quarters. This time includes breaks, rest periods, and meals, but not time away from work necessitated by the study procedures. Again, no significant difference in the frequency or duration of rest periods was noted in comparing the cardiac workers with their controls.

The specific type of heart disease showed no influence on the amount or pattern of energy expenditure when paired subjects and controls were compared within diagnostic categories. In the largest category, that of arteriosclerotic heart disease with previous myocardial infarction, the average rate of energy expenditure was identical for the 23 patients and 17 controls, namely 1.94 Calories per minute. The other diagnostic categories were small (one to five pairs), so that comparisons within them are of limited value.

PHYSIOLOGIC PARAMETERS OF CARDIOVASCULAR FUNCTION

Clinical observations and physiologic data were analyzed for evidence of stress that might distinguish the response of the subject with heart disease from that of his healthy co-worker, or that might be considered injurious to the cardiac patient.

Blood Pressure

The average systolic blood pressure at rest and at work was higher in the cardiac

group than among the controls, but the diastolic blood pressures were nearly identical. The differences in systolic pressure were statistically significant and were probably the result of the significantly greater age of the group with heart disease, rather than the inclusion of patients with hypertension since there were only three of the latter (Table 14-3).

TABLE 14-3 AGE AND BLOOD PRESSURE

	36 Cardiacs		26 Controls		
	Avr- age	S.D.	Avr- age	S.D.	P
Age (years)	50.6	9.4	41.5	12.3	< .02
Systolic blood pressure at rest (mm. Hg)	135	22	122	19	< .05
Diastolic blood pressure at rest (mm. Hg)	81	10	81	13	—
Maximum systolic blood pressure at work (mm. Hg)	153	29	134	20	< .02
Maximum diastolic blood pressure at work (mm. Hg)	89	10	88	13	—

None of the cardiacs developed a working blood pressure higher than 184/100 mm Hg except for two patients with hypertension and two with aortic insufficiency.

It may be noteworthy that the subject whose blood pressure rose to 184/100 mm Hg at work was a 62-year-old man who had previously recovered from a myocardial infarction and who died suddenly 3 months after the study. None of the control subjects developed a working blood pressure higher than 162/108 mm Hg except for three who were found to have previously unrecognized hypertension at rest (diastolic pressure over 100 mm Hg).

The average maximum increase of blood pressure was 17 mm Hg systolic and 8 mm Hg diastolic for the cardiacs and slightly less for the controls (Table 13-3).

The highest blood pressures recorded during the working day coincided with maximum energy expenditure in only 26 per cent of the total cases and in only one of 13 subjects, whose blood pressure during work rose above 150 mm Hg systolic or 100 mm Hg diastolic. During performance of factory work in the study, therefore, the blood pres-

sure response elicited was small in the normotensive cardiacs and controls and probably not harmful. In known or unrecognized hypertensive subjects, however, although the increment was similarly small, it is possible that the levels reached in certain subjects might have structural or functional significance.

Pulse Rate

The average pulse rate increased from 84 per minute at rest to an average maximum of 102 per minute during work. Sinus tachycardia (over 110 per minute) occurred in 17 subjects, 10 controls and 7 cardiacs. In 7 subjects the sinus rate was 120 per minute or more. Three were control subjects, all of whom had resting pulse rates over 100 per minute; two had previously undetected hypertension, and the third was obese. Two of the four cardiac subjects had high resting pulse rates. A third subject developed the highest observed pulse rate (136 per minute). He was a 59-year-old man with rheumatic heart disease and predominant aortic stenosis, classified II C. This subject died suddenly four months after the study. The marked tachycardias were disproportionate to the rate of energy expenditure, since they occurred at rates of 1.8 to 4.3 Calories per minute. In general, there was poor correlation between pulse rate and energy expenditure at the relatively low rates of this study. Even at the highest levels studied, the maximum pulse rate coincided with the maximum rate of energy expenditure in only 35 per cent of the cases. This type of factory work, therefore, elicits only a moderate increment in pulse rate. Under the conditions described, a working pulse rate in excess of 120 per minute suggests an undetected or inadequately treated cardiovascular abnormality, sustained anxiety, or limited cardiovascular reserve.

Respiratory Rate

The average respiratory rate in the cardiac group was 16.4 per minute at rest and 22.7 per minute during maximum exertion,

while the average pulmonary ventilation rose from 8.7 liters per minute to 16.9 liters per minute. The figures were slightly higher for the control group, but the difference was not statistically significant when matched pairs were compared. The highest individual values for respiratory rate and pulmonary ventilation were correlated with high rates of energy expenditure rather than the specific cardiovascular abnormalities noted in association with high blood pressure and pulse rate.

Oxygen Utilization

Oxygen utilization is the number of milliliters of oxygen removed from each 100 ml of air breathed, and equals ten times the reciprocal of ventilatory equivalent. Oxygen utilization normally rises with exercise. In the cardiac group it increased from 3.10 ml of oxygen per 100 ml of air at rest to 3.85 ml of oxygen per 100 ml of air. Both figures were slightly higher in the control group, but the difference was not significant. As with pulmonary ventilation, high individual values occurred during high rates of energy expenditure.

Electrocardiographic Changes

There were remarkably few electrocardiographic changes. The method of electrode placement yielded information only in the horizontal plane, i.e., on the x and z axis, equivalent to RV_8 , V_4 , and V_6 .

In the cardiac group, there were two patients with atrial fibrillation, four with ventricular premature beats, and three cases of atrioventricular block (one, prolonged P-R interval, one, second degree A-V block with Wenckebach phenomenon, one, complete A-V block).

During work, the premature beats persisted but did not increase in frequency; in four additional cases, rare ventricular premature beats appeared. Three of the four subjects had shown an abnormal response to the Master exercise tolerance test performed previously. The premature beats did not appear with maximal energy expenditure. Three subjects showed primary T

wave changes (change in angle QRS-T) during the working day. Two of the three subjects also had abnormal blood pressure response. One of the two mentioned above had a maximum abnormal pulse rate response, and died later at home in bed.

In the subject with second degree A-V block (Wenckebach phenomenon), ventricular premature beats appeared and the phenomenon became more prominent. In the subject with third degree A-V block the pulse rate increased from 56 to a maximum of 58 per minute although the energy expenditure ranged from 187 to 249 Calories per minute.

The control subjects all had regular sinus rhythm. Two subjects had ventricular premature beats at rest that disappeared in one individual during work. Primary ST-T wave changes occurred in three cases at levels of energy expenditure of 18 to 28 Calories per minute.

The electrocardiographic changes in cardiac and control groups were similar except for the appearance of ventricular premature beats, which during work occurred only in the cardiac group. The low incidence of ST-T wave changes at this level of work in these subjects is not surprising since only 23 per cent had shown abnormal or borderline response to Master's exercise tolerance test, which requires larger expenditures of energy.

DISCUSSION

How representative are the jobs studied? Although not selected for statistical purposes, the jobs appear to be typical of those held by a third of the total working population of a large industrial city, and by a large percentage of working cardinals studied at The Work Classification Clinic of The Cleveland Area Heart Society. The jobs ranged from mental to managerial and from sedentary to active, and were distributed throughout all departments of these three metal manufacturing plants.

The energy cost of the type of work studied is remarkably low in relation to

other human activities. A champion athlete can maintain an energy expenditure of 265 Calories per minute for several minutes during a two-mile run or skiing.^{12,14} Taylor, Buskirk, and Henschel¹⁵ found that a group of healthy but untrained young men could achieve a maximum rate of energy expenditure of approximately 18 Calories per minute on a treadmill. These rates are obviously unrealistic in relation to the requirements of daily work. Passmore and Durnin,⁴ reasoning from their own data and those of German workers, conclude that 5 Calories per minute during working hours "probably represents the upper rates of daily energy expenditure that can be maintained regularly in heavy industry." Garry and his associates,¹⁶ using methods similar to those of the present study, found the average rate of energy expenditure by coal miners during the underground shift to be 4.3 Calories per minute. In the same study, clerks at the mine had an average working rate of 1.7 Calories per minute. In the present study, the average rate of energy expenditure was more comparable to the latter group. During the working shift for the entire group of cardinals, the rate was 1.97 Calories per minute, ranging from 1.54 Calories per minute for the miscellaneous group (planning clerk, tool and laundry crib attendant, etc.) to 2.35 Calories per minute for the warehousemen (stock pickers) (Fig. 14-1). The energy requirements of these jobs, therefore, lie in the lower range of possible sustained energy output. In fact, the workers may expend energy on the job at a lower rate than they do when not working.¹ This surprising conclusion is based on the findings of Garry and his co-workers¹⁶ that, during the eight nonworking, waking hours, 1400 Calories or 2.96 Calories per minute were expended by coal miners and clerks, a rate that is higher than the average working rate for any of the groups in the present study. The low rates of energy expenditure at work are not attributable to heart disease as such, since no statistically significant difference in either average or maximum rate could be demonstrated when the cardinals

were compared with healthy men performing the same job. The subjects had not been downgraded to less difficult jobs because of developing heart disease, according to the industrial physicians, consulting physicians (Work Classification Clinic of Cleveland Area Heart Society), and personnel managers. Furthermore, there were cardiac workers in all departments and types of work in these industries with the single exception of nonferrous metal casting, where excessive heat may impose a serious burden on the cardiovascular system.¹⁷

Granted, then, that the energy demands of such factory work are not great, does the work nevertheless impose dangerous stress on the cardiovascular system of workers with heart disease? Certain individuals have been cited above who, in spite of the relatively light character of the work, developed heart rates of over 120 per minute or blood pressures of over 184 systolic or 90 diastolic, and electrocardiographic changes. These physiological alterations may have variable significance. When they occur singly and infrequently, as in the control group, they may be insignificant or secondary to obesity or benign essential hypertension. The concurrence of several abnormalities in the same subject is probably significant. Their occurrence in two subjects who died unexpectedly within four months of the study, however, suggests that they represent an encroachment upon a diminished cardiac reserve. It would be desirable, although not presently practicable, to monitor several parameters of cardiovascular function in the average cardiac at work, in order to detect such evidence of stress. Standardized exercises such as the Master Two-step Test may be used to elicit such changes in the office or laboratory. It is important to recognize, however, that the Master step test calls for the expenditure of 8.5 Calories per minute, a rate in excess of any peak observed in any of the workers studied.² Certainly, an individual with heart disease who can make this exertion without developing excessive heart rate, blood pressure, or abnormal electrocardiographic changes would not be expected to show adverse effects during a job

requiring average peaks of 3.45 Calories per minute, or average sustained effort of only 2 Calories per minute. The cardiac who does show an abnormal step test response may, nevertheless, be able to perform at the rates of energy expenditure described. The decision to return such a cardiac to work may be simplified by the realization that merely keeping him at home without further restriction may not reduce his energy expenditure below that required by work. The patient's work may, of course, entail forms of stress other than those measured in calories, but emotional and personal problems may be aggravated, and are seldom solved by keeping a worker idle.

The application of energy studies to the problems of heart disease is a new field. Considerable data exist but deserve wider application.⁴ The energy requirements of additional types of activity (agricultural, industrial, domestic, etc.) are needed for better vocational counseling of the patient with heart disease. More extensive surveys will be possible in the future with automatic equipment to record pulmonary ventilation. The use of pulmonary ventilation as an index of energy expenditure has been suggested by Durnin and Edwards,¹⁸ and confirmed and extended by analysis of the data of the present study reported elsewhere.¹⁹ In work more strenuous than that studied here, it may be possible to use energy studies to delineate the pattern of energy expenditure, and eliminate or redistribute peaks of exertion in order to adapt the job better to the worker.

Two major problems persist to which the solutions are not readily apparent. The first is the definition and measurement of the types of stress other than simple energy expenditure that make a job *hard*. The second is the definition and measurement of fatigue. It is unlikely that any of the workers in this study could have been physically exhausted by the expenditure of a thousand calories during an 8½ hour shift. Yet many were undeniably *tired*. The physiological basis of this symptoms in the cardiac and its relation to disturbed cardiovascular function constitute an important field for future

investigation, quite apart from the study of fatigue in the normal subject

SUMMARY

Sixty-two subjects, 36 with heart disease and 26 healthy controls, were observed during a normal working day in three metal manufacturing plants. The workers are considered representative of at least a third of the working population of a large industrial city. Their average rate of energy expenditure during the shift was of the order of 2 Calories per minute, a low rate, comparable to that observed during nonworking activities. The maximum rate of energy expenditure rarely exceeded twice the resting rate. A few subjects with heart disease developed disorders of cardiovascular function in terms of the blood pressure, heart rate, or electrocardiogram that may have had serious prognostic significance.

Modern factory work of the type studied requires low rates of energy expenditure and is well within the capacity of a patient with heart disease who is not acutely ill or severely decompensated.

BIBLIOGRAPHY

- 1 HELLERSTEIN, H. K., and FORD, A. B. The rehabilitation of the cardiac patient, *JAMA* 164 225, 1957.
- 2 FORD, A. B., and HELLERSTEIN, H. K. The energy cost of the Master two-step test, *JAMA* 164 1868, 1957.
- 3 BRIDGE, R. A., LOVEJOY, F. W., JR., YU, P. N. G., and McDOWELL, M. E. Evaluation and significance of physical fitness for moderate work, study of patients with cardiovascular or pulmonary diseases, *AMA Arch Indust Hyg* 4 236, 1951.
- 4 PASSMORE, R., and DURNIN, J. V. G. A. Human energy expenditure. *Physiol Rev* 35 801, 1955.
- 5 NEW YORK HEART ASSOCIATION, INC. *Nomenclature and Criteria for Diagnosis of the Heart and Blood Vessels*, by Criteria Committee, HAROLD E. B. PARDEE, Chairman. 5th ed. New York, Am Heart Assn, 1953.
- 6 *Occupational Inventory of the Number and Kinds of Jobs in Cuyahoga County, 1946*. Occupational Planning Committee of the Welfare Federation of Cleveland, 1947.
- 7 MILLER, E. A., and FRANZ, H. Energieverbrauchs-messungen bei beruflicher Arbeit mit einer verbesserten Respirations-Gasuhr, *Arbeitsphysiol* 11 499, 1952.
- 8 SCHOLANDER, P. F. Analyzer for accurate estimation of respiratory gases in one-half cubic centimeter samples, *J Biol Chem* 167 235, 1957.
- 9 CATHCART, E. P., and CUTHBERTSON, D. P. The composition and distribution of the fatty substances of the human subject, *J Physiol* 72 349, 1931.
- 10 TANNER, J. M. Fallacy of per-weight and per-surface area standards and their relation to spurious correlation, *J Appl Physiol* 21, 1949.
- 11 MAHADEVA, K., PASSMORE, R., and WOOLF, B. Individual variations in the metabolic cost of standardized exercises: the effects of food, age, sex and race, *J Physiol* 121-225, 1953.
- 12 SNEDECOR, G. W. *Statistical Methods as Applied to Experiments in Agriculture and Biology*. 4th ed. Ames, Ia., The Collegiate Press, 1946.
- 13 CHRISTENSEN, E. H., and HÖGBERG, P. Physiology of skiing, *Arbeitsphysiologie* 14 292, 1950.
- 14 ROBINSON, S., EDWARDS, H. T., and DILL, D. B. New records in human power, *Science* 85 409, 1937.
- 15 TAYLOR, H. L., BUSKIRK, E., and HENSCHEL, A. Maximal oxygen intake as an objective measure of cardio-respiratory performance, *J Appl Physiol* 8-73, 1955.
- 16 GARRY, R. C., PASSMORE, R., WARNOCK, G. M., and DURNIN, J. V. G. A. *Studies on Expenditure of Energy and Consumption of Food by Miners and Clerks, Fife, Scotland, 1952*. Medical Research Council Special Report Series No. 289. London, Her Majesty's Stationery Office, 1955.
- 17 CHRISTENSEN, E. H. "Physiological Valuation of Work in Nykroppa Iron Works," in FLOYD, W. F., and WELFORD, A. T. (eds.), *The Ergonomics Research Society Symposium on Fatigue*, March, 1952. London, H. K. Lewis & Co., 1953, pp. 93-108.
- 18 DURNIN, J. V. G. A., and EDWARDS, R. G. Pulmonary ventilation as an index of energy expenditure, *Quart J Exper. Physiol* 40 370, 1955.
- 19 FORD, A. B., and HELLERSTEIN, H. K. Pulmonary ventilation as an index of energy expenditure, *Clin Res Proc* 3 227, 1955.

CHAPTER 15 THE MEASUREMENT OF CARDIAC PERFORMANCE IN OSTENSIBLY WELL, OLDER INDIVIDUALS

Milton Landowne

In the selection of effective tests of cardiovascular performance for use in employment of persons with cardiac disease, it is valuable to compare results obtained in known and suspected cardiac patients with results obtained in ostensibly well, working subjects, as well as in the rarer subject with ideal or optimal function. The more specific measures are not readily available for this purpose. Some procedures present serious technical difficulties to wide application. In other specialized procedures the indications do not always outweigh the estimated inconvenience and risk and the use of these methods is limited to selected material. On the other hand, measures that are convenient and accurate may have little specificity and their interpretation in quantitative terms is limited. Such indexes may be useful for group evaluation but in an individual instance they are open to valid criticism.

Our laboratory has been interested in the

description of physiological behavior of subjects who are characterized primarily as to chronologic age and secondarily as clinically free of disease pertinent to the function of system under study. The interpretation of our findings on ostensibly well subjects will in some measure depend upon the influence of what may be called *impertinent disease*—that which is clinically unrecognized or unevaluated. Because the influence of subclinical functional or structural disease may be a dominating agewise variable in the circulation of ostensibly well persons, I have been particularly interested in the specificity, sensitivity, and reliability of measures used to evaluate cardiovascular function. I shall try to invite your discussion in three related areas. These are (1) the estimation of cardiovascular performance at rest, (2) the use of expressions for prediction or determinations of cardiac output from blood pressure data, (3) the evaluation of cardiovascular performance during exercise.

ESTIMATION OF CARDIOVASCULAR PERFORMANCE AT REST

When cardiac output was measured in a group of ostensibly well males by Dr Brandfonbrener,¹ we noted a moderate scatter in the values. This was shown by the standard deviation of distribution, which was about one third of the mean value (Fig. 15-1). Technical variability of the dye dilution method used accounted for very little of this. Body size and heart rate variation explained some of the differences between individuals, and a systematic decrease in stroke index with age accounted for part of the remaining variability (Fig. 15-2). Describing this age-wise relation does not explain it. It could be argued that the age-wise decrease in output was due to subclinical cardiovascular disease or to functional impairment. This is a good possibility since it is likely that the incidence and the severity of cardiovascular disease increase with age even more for the unrecognized than for the clinically recognized forms. Lacking a primary standard of optimal cardiovascular function, we used two additional relations to judge the capability or adequacy of performance.²

The age-wise increase in the relationship of pressure to flow, expressed as peripheral resistance, adequately explains a declining output as the simple consequence of a decreasing vascular bed. On the other hand, the reduction in output results in a reduction in calculated left ventricular work at rest and would seem to represent the reduced response to a reduced demand. Left ventricular work was estimated as the product of stroke volume and average systolic pressure. The latter was obtained from the area under the brachial pressure curve during systole. Systolic termination was judged as the point of most rapid pressure decline after the systolic peak. When stroke work was expressed in relation to heart size, the variability (28 per cent) was no greater than that noted for a critically studied series of young normals.³

However, cardiac size was not decreased

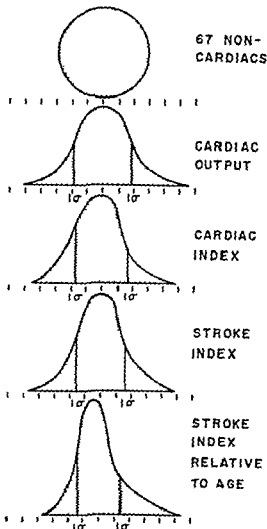


FIG. 15-1 Among an apparently homogeneous group of normals, the resting cardiac output varies considerably ($\sigma = 32$ per cent). This dispersion is progressively reduced when account is taken of differences in body size (cardiac index), of heart rate (stroke index), and of age (stroke index relative to age). A good deal remains, to be called individual variability ($\sigma = 23$ per cent), despite these attempts to narrow the categorical limits of normal (Data from Brandfonbrener, Landowne, and Shock¹).

with age, therefore work diminished relative to cardiac size in some subjects. In addition, systolic duration increased and, in consequence, the rate of stroke work or power decreased with age. These indexes, work/heart size, and power not only indicate a decrease in the performance of the left

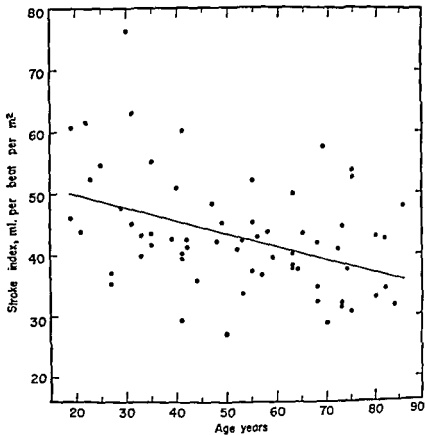


FIG 15-2. Relation of resting stroke index and age for 67 clinically well males (From Brandfonbrener, Landowne, and Shock,¹ reproduced by permission).

ventricle with age but, unless the end diastolic left ventricular pressure were reduced, which is unlikely, they indicate a decrease in reserve or capability as well.

The above analysis shows an agewise decrease both in capability and in circulatory requirement in the same material. Even if this is appropriate to the group, it does not follow that the individual characterization of work/heart size or of *power* is a suitable estimate of individual cardiac ability. Some other measure of cardiac reserve should serve as a standard for individual comparison and the cardiac response to exercise has been suggested for this purpose

DETERMINATION OF CARDIAC OUTPUT FROM BLOOD PRESSURE DATA

Because the dye dilution method limits the number of output determinations that

can be made in a subject during an exercise period, we have examined several other means of obtaining measures of cardiac output. The relation between blood pressure and flow is essentially determined by the properties of resistance and distensibility of the vascular bed. If the latter were known, the flow or output might be derived from the blood pressure. In our study of resting output² we had calculated a value for distensibility (E). This value provided us with the encouragingly high age correlation coefficient of $+0.76$. This correlation is statistically highly significant. If it had been perfect, a prediction of individual arterial distensibility could be made accurately from the individual's age. The accuracy of this prediction depends upon the variability that is not age-dependent. This is shown by Figure 15-3, which is a plot of the correlation coefficient (r) against $\sqrt{1-r^2}$, the

fraction of the standard deviation of the group that is independent of the correlation. For a correlation coefficient of .98 the standard deviation about regression, a measure of the age independent variability, would be 20 per cent of the standard deviation, if the group were considered without regard to age. For a correlation coefficient of .76 the age independent standard deviation would be nearly two thirds of the standard deviation of the group. And if cardiac output were calculated from the blood pressure using the distensibility predicted from the subjects age, the same uncertainty would be present. In this case only half of the individuals would have predicted values within ± 20 per cent of the measured values.

The statistical fact that the standard deviation about regression remains sizable even at high correlation coefficients is thus one of the limitations to individualizing information obtained for correlation data. Since empirical formulas for cardiac output are totally dependent upon correlation information, individual prediction may frequently be inaccurate. For example, one of the expressions proposed by Starr for the prediction of stroke volume from blood pressure and age⁴ was tested on our material and revealed considerable variable as well as absolute discrepancy. The error of prediction was greater than ± 20 per cent for 63 per cent of the subjects. The average discrepancy was 26 per cent but this can be reduced by adjusting the constant term of the formula. The standard deviation of the discrepancy between predicted and measured stroke volume was 24 per cent of the observed value. Adjusting the constant term to eliminate the average error would still leave an error of greater than 20 per cent in 40 per cent of subjects. In less homogeneous material, even greater variability in discrepancy would be anticipated.

The simplest basis for several empirical formulas considers pulse pressure to be proportional to stroke volume. This ratio increases significantly with age, in our material the error of estimation of stroke

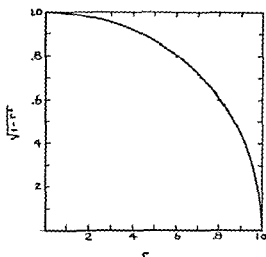


FIG 15-3 Relation between the correlation coefficient (r) and the fraction of the standard deviation of the distribution which is independent of correlation ($\sqrt{1-r^2}$)

volume from age for this simple expression would be expected to exceed 20 per cent in 56 per cent of subjects.

We next considered whether individually determined parameters might be used to interpolate cardiac output between two determinations and examined the factors relating pressure and flow more closely. The expression for arterial distensibility (E) was based on a simplification of the familiar model that assumes a constant central distensibility (E) and peripheral resistance (R) during diastole. Under these conditions the diastolic fall in blood pressure would be exponential, at a fractional or logarithmic rate determined by E/R , the ratio of distensibility to resistance. The simplification used was to consider the pressure fall as linear and to obtain the desired fractional rate from the average rate of diastolic pressure fall divided by the mean diastolic pressure. The product of E/R and the mean blood pressure is the expression EF , where F is flow, since R is the ratio of pressure to flow. At rest E would be expected to be constant, while the observed variability of F was about 6 per cent. EF was calculated from blood pressure curves for 54 replicate pairs. The standard deviation of replicates was 11 per cent, indicating a

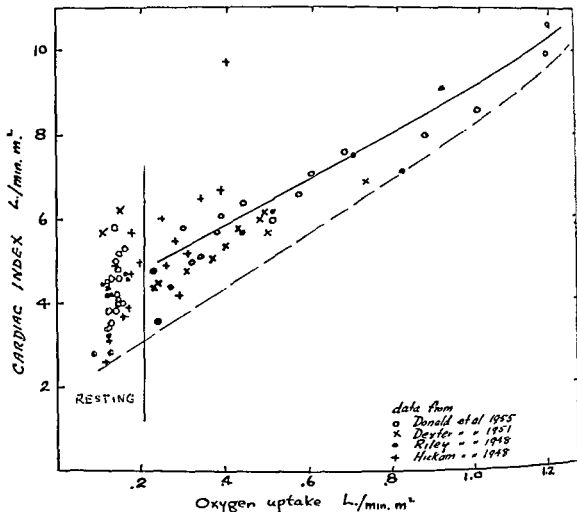


FIG 15-4 Relation between cardiac index and oxygen uptake at rest and during exercise in normal subjects. Composite data from several sources. Solid regression line is for exercise data of Donald *et al*. Dashed line describes lower limit of data.

good degree of reproducibility of E by this method.

We, therefore, were encouraged to examine the diastolic pressure curve and the expressions used more closely. Under resting conditions, the degree of approximation of diastolic pressure curves to an exponential form was examined by curve-fitting techniques applied to enlarged accurately obtained records of brachial, femoral, subclavian, and aortic pressures. An equation of the type $p = p_0 e^{-\lambda t} + p_a$ provided an excellent description of diastole in most trials, the standard deviation about the fitted curve was often less than 1 mm Hg.⁵

Seven expressions relating output to the

rate of diastolic pressure fall were examined and tested for reproducibility. There was little difference among them. The theoretically preferable expression was then tested on a series of 19 subjects. EF was obtained twice for each subject from the average of three beats each. These pairs showed a standard deviation of 14 per cent. In a series of six subjects, replicate cardiac output determinations provided a measure of F . The discrepancy between predicted second value of F and the observed flow ranged from -29 to 8 per cent. Attempts were made to apply these expressions to exercising conditions but were discouraging. During exercise, diastolic pressure curves are not satisfactorily de-

scribed by simple exponential functions and beat-to-beat variation is marked. The expressions used to estimate stroke volume from blood pressure at rest become inapplicable during exercise in our hands.

CARDIOVASCULAR PERFORMANCE DURING EXERCISE

Earlier observations⁶ that the increase in cardiac output during muscular exercise was proportional to the accompanying increase in oxygen uptake have been amply corroborated by succeeding studies with newer techniques. This proportionality has been proposed as a measure of the cardiac response to exercise. Resting variability, including the nonbasal state, and the rela-

tively small increases in oxygen uptake at low rates of exercise, make determination of the ratio (increase in output/increase in oxygen uptake) somewhat variable. Donald and his associates⁷ used the information from a group of subjects during exercise to construct a regression of cardiac index on oxygen uptake during exercise. Figure 15-4 presents composite data to show this regression, and its relation to resting values and to the data of others.

The extraction of oxygen from blood by working muscle is far more complete than for the average body tissues at rest, therefore exercise leads to an increase in A-V difference for the body as a whole. Let us roughly consider the circulation and oxygen uptake during exercise as if they were sep-

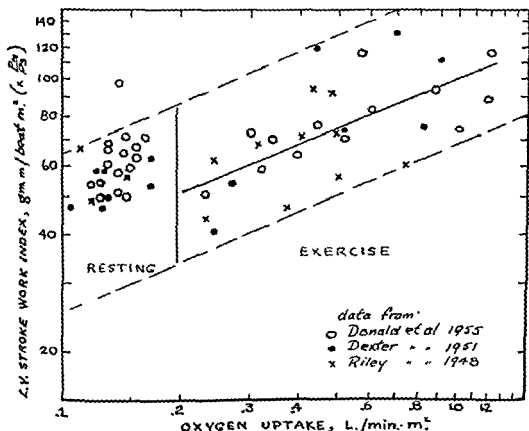


FIG 15-5 Relation between left ventricular stroke work index and oxygen uptake at rest during exercise in 30 normal subjects. Composite data from several sources. Solid regression line represents exercise data as $\text{Stroke work index (gm m/beat m}^2\text{)} = 100 \text{ O}_{2\text{ uptake (L/min m}^2\text{)}}$. Dashed lines enclose all but one observation.

arated into two areas of supply, one to the exercising or "working" area, and the other to the resting or nonworking remainder. The increase in oxygen uptake represents the oxygen used to meet the needs of muscular activity; it parallels roughly the amount of muscular activity. The increase in cardiac output will supply part or all of this oxygen increase to the muscles. The ratio $\frac{\text{increase in oxygen}}{\text{increase in output}}$ therefore reflects a hypothetical A-V difference in the working area. The average change in output observed by Donald and his co-workers during exercise was 5.34 ml. blood per milliliter change in oxygen. Expressed as the reciprocal, the average "A-V difference in the working area" was 187 volumes per cent. It is unlikely that the average desaturation of arte-

rial blood in the working area is as complete as this and diversion of blood from abdominal organs is known to accompany muscular exercise. However, the assumption of complete unsaturation of blood provides an estimate of the *minimal* diversion of blood from nonworking to working areas. Estimates of minimal diversion in relation to the cardiac output and blood pressure provide approximations of the minimal vascular resistance of the nonworking area. This affords a perspective of the adjustments to exercise different from that evident from the consideration of the total mixed A-V difference; it is an interpretative way of expressing the same information. Where arterial oxygen content is low or the oxygen cost of work is increased, adjustment calls for a greater increase in output or a greater

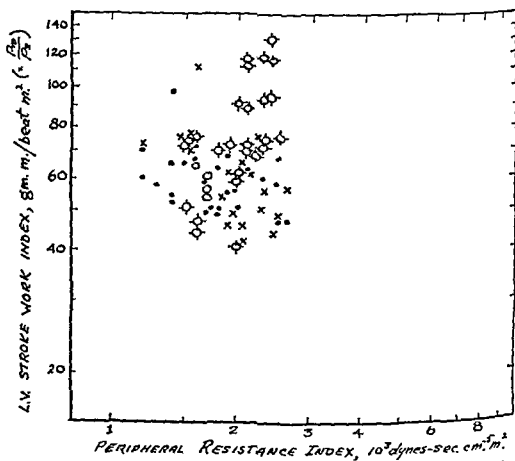


FIG. 15-6. Relation between left ventricular stroke work index and "nonworking" peripheral resistance index in normal subjects. Large symbols refer to exercising subjects, and dots to resting subjects from Fig. 15-5. Crosses represent 18 additional resting subjects aged 19 to 39 years.

diversion of blood from nonworking areas or both. One may consider that a competent heart delivers an optimal increase in output, so that flow and pressure are adequately maintained. Under these conditions the diversion of blood from nonworking areas is minimized and the nonworking resistance change is minimal. Where cardiac output is not adequate to supply oxygen for the maintenance of steady state work, a greater circulatory diversion is demanded. Greater peripheral constriction adds to the burden of cardiac work in a more costly manner than does greater cardiac output.

Just as in the resting state, the stroke work index may furnish a more useful measure of left ventricular performance during exercise than does the cardiac index. The peripheral diversion is reflected in the estimate of nonworking resistance. Figure 15-5 indicates the regression and scatter of left ventricular stroke work index for most of the material presented in Figure 15-4. Figure 15-6 relates this stroke work index to the peripheral resistance index under resting conditions and to the "nonworking" resistance during exercise. Our resting values for 18 subjects under the age of 40 are included. It is suggested that this relationship may be useful in the estimation of central and peripheral cardiovascular performance at rest and during muscular exercise.

BIBLIOGRAPHY

- 1 BRANDFONBRENER, M., LANDOWNE, M., and SHOCK, N. W. Changes in cardiac output with age, *Circulation* 12 557, 1955.
- 2 LANDOWNE, M., BRANDFONBRENER, M., and SHOCK, N. W. The relation of age to certain measures of performance of the heart and circulation, *Circulation* 12 567, 1955.
- 3 STARR, I., DONALD, J. S., JR., MARGOLIES, A., SHAW, R., COLLINS, L. H., and GAMBLE, C. J. Studies of heart and circulation in disease, estimations of basal cardiac output, metabolism, heart size, and blood pressure in 235 subjects, *J Clin Invest* 13 561, 1934.
- 4 STARR, I., SCHNABEL, T. G., JR., ASKOVITZ, S. I., and SCHILD, A. Studies made by simulating systole at necropsy, on relation between pulse pressure and cardiac stroke volume, leading to clinical method of estimating cardiac output from blood pressure and age, *Circulation* 9 648, 1954.
- 5 LANDOWNE, M. *Analysis of Diastolic Blood Pressure*, XXth International Physiol Congr Brussels, 1956, p 544. (Abstr. Communications.)
- 6 BAINBRIDGE, F. A. *Physiology of Muscular Exercise*, 3d ed., rewritten by A. V. BOCK and D. B. DILL. London, Longmans, Green & Co, 1931.
- 7 DONALD, K. W., BISHOP, J. M., CUMMING, G., and WADE, O. L. The effect of exercise on the cardiac output and circulatory dynamics of normal subjects, *Clin Sc* 14 37, 1955.

CHAPTER 16 EVALUATION OF FUNCTIONAL CAPACITY IN PATIENTS WITH CARDIOVASCULAR DISEASE*

Robert A. Bruce, Donal R. Sparkman, Robert M. Levenson, and Albert M. Hurley

Functional capacity has been defined as "an estimate of what the patient's heart will allow him to do."¹ Physiologically, it varies with the natural history of heart disease and the effects of treatment. Clinically, it varies with circumstances affecting the awareness of symptoms in relation to energy requirements of customary activities deemed ordinary by the patient. Furthermore, the cardinal symptoms of dyspnea, orthopnea, chest pain, fatigue, or palpitation are not specific, and must be differentiated from emotional reactions and symptoms of other diseases. Cardiac patients often have other nonspecific symptoms.

Classification

Functional capacity has been divided, by the New York Heart Association, into four

* This investigation was supported by grants from the Washington State Heart Association and the National Heart Institute, Department of Health, Education, and Welfare.

categories (Table 16-1).² Thus, despite physical manifestations of organic heart disease, Class I patients are asymptomatic during "ordinary" activity. Class II patients sometimes notice either symptoms, limitations, or both with such activity. Class III patients develop symptoms and find themselves limited, with less than ordinary activity. Class IV patients may have symptoms with the slightest effort, and often at rest. Indeed, they are limited to virtually a bed-chair existence.

Determination

Although a complete history and physical examination are necessary to diagnose heart disease and to differentiate it from other possible causes of illness, the functional capacity is derived largely from historical information. Usually, in the absence of cardiomegaly, signs of heart failure, or arrhythmias, physical examination is of little help

TABLE 16-1. CLINICAL EVALUATION OF FUNCTIONAL CAPACITY OF PATIENTS WITH HEART DISEASE IN RELATION TO ORDINARY PHYSICAL ACTIVITY
(Modified from New York Heart Association, 1953)

Class	Cardiac symptoms	Limitations	Need for additional rest*	Physical ability to work†
I None		None	None	Full time
II Only moderate or occasional		Slight	Usually only slight or occasional	Usually full time
III Definite, with less than ordinary activity		Marked	Usually moderate	Usually part time
IV May be present even at rest, and any activity increases discomfort		Extreme	Marked	Unable to work

* To control or relieve symptoms, as determined by the patient rather than advised by the physician

† At accustomed occupation or usual tasks

in this regard. Evaluation depends upon the patient's description of his symptoms with respect to the circumstances, severity, and frequency of common types of exertion, such as walking on the level at a customary pace, walking up a familiar hill, or climbing a flight of stairs. It is also pertinent to inquire how far the patient can walk, or how many flights of stairs he can climb, before noticing symptoms. How long do these symptoms persist on stopping the effort? Are there other contributory factors that are known to cause exertional symptoms, such as excitement, eating a heavy meal, or exposure to cold? Does either frequency or severity of these symptoms fluctuate over a period of time? Does a restful week end change the symptomatology? Do symptoms occur during sleep, and if so, are they relieved by sitting up?

Because of the numerous variables that make differentiation of the intermediate gradations of functional capacity difficult, two additional criteria are helpful. One is the need for additional rest, as determined spontaneously by the patient (Table 16-1), and the other is the physical ability to perform the customary activities on a regular schedule without self-imposed limitations.² Class II patients usually do not have much need for additional rest, or at least do not shorten their working hours to achieve it. Instead, if need be, they obtain more rest off the job, such as during evenings and week ends, and may thereby curtail their usual recreational activities. Class III patients, despite a strong motivation to con-

tinue ordinary activities, usually find it physically impossible, and must, therefore, shorten working hours in order to obtain more rest.

Limitations

Actually, the classification of a particular patient may also vary between Class II and III categories, depending upon the customary level of energy requirements involved in the daily activities. Thus, with a low-energy requirement a patient may be able to work virtually full time, whereas with a higher requirement he would be unable to work more than part time.

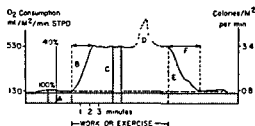
In addition, other physical illness, neuropsychiatric illness, poor motivation, or socioeconomic factors may affect the subjective awareness of symptoms. Finally, even when functional capacity is determined, it applies only to the status at the moment, and is subject to change in accord with the natural history of disease and its treatment.

Energy Requirements for Ordinary Activities

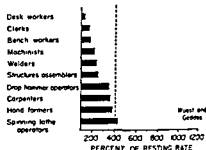
Energy requirements may be assessed from the approximate rate and duration of excess oxygen consumed in performance of effort. Although other characteristics, such as duration of time to achieve a steady state, oxygen debt, and recovery time are informative, the simplest measure* of intensity of effort is the percentage ratio of oxygen consumption at work compared to that at rest. This value fluctuates with work require-

* This eliminates corrections for age, sex, and body size as well as other variables such as anxiety, heat stress, etc.

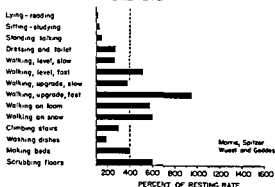
ANALYSIS OF ENERGY REQUIREMENTS



APPROXIMATE INTENSITY OF ENERGY REQUIREMENTS IN INDUSTRY



APPROXIMATE INTENSITY OF ENERGY REQUIREMENTS IN DAILY LIFE



APPROXIMATE INTENSITY OF ENERGY REQUIREMENTS ON THE FARM

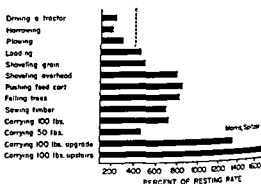


FIG 16-1 Analysis of energy requirements by means of graphic recording of oxygen consumption. *A* represents requirement at rest; *B*, lag in increase of oxygen consumption with work or exercise; *C*, average value per minute during a steady state of physical activity; *D*, further increment with peak load; *E*, oxygen debt; and *F*, time lag for recovery.

The approximate intensity of energy requirements for representative activities in daily life, industrial jobs and work on the farm are expressed as percentages of resting requirement, corresponding to value *C*, above.

ments, increasing with peak loads to higher levels.

The approximate energy requirements of ordinary daily activities may be placed within broad categories representing slight, moderate, severe, and very severe work. Thus sitting, standing, or talking represent only slight physical activities, whereas walking slowly on the level requires an appreciable though only moderate energy expenditure (Fig. 16-1). Walking upgrade on a smooth, hard surface or walking on the level on loam or snow requires greater energy expenditures, representing severe work.³ Walking upgrade on uneven terrain at a rapid pace, i.e., hill climbing, is severe work.

Similar measurements of approximate energy requirements of common jobs in an aircraft industry have been made available for comparison.⁴ Since much of the work is more or less mechanized, the energy expenditure for the particular jobs sampled does not seem to represent very severe work. In like manner the energy requirements on the farm indicate corresponding ranges for mechanized tasks, plus much higher energy requirements for certain manual tasks and chores.^{3,5} These data do not take into account environmental heat stresses that often occur.

Wuest and Geddes did not find any significant difference in the energy require-

ments for noncardiac and cardiac employees performing the same industrial jobs.⁴ A slightly lower oxygen consumption during work in some cardiacs was attributed to avoidance of the more strenuous aspects of work, but the possibility of slightly greater efficiency owing to longer experience on the job could not be excluded.

CLINICAL INVESTIGATION

Ninety-six patients with coronary artery disease have been studied with respect to

the facts, appraisal of the functional capacity, and recommendation of the work prescription (Tables 16-2, 16-3). This test, which involves large muscle masses and requires no training, is standardized, as described elsewhere, by having an ambulatory patient walk on a motor-driven treadmill at

TABLE 16-3. EXERCISE RESPONSES WITH CORONARY ARTERY DISEASE

Functional capacity Number of patients	I 16	II 52	III 29	All 96
Anxiety	1	26	19	46
Dyspnea	5	15	25	65
Chest pain	0	19	15	34
Fatigue	2	12	15	29
Endurance,* minutes	10 ± 0	8 0 ± 2 6	6 0 ± 3 2	
Hypotension	0	1	3	4
Systolic rebound	0	0	3	3
VPS	0	3	2	5
ST	1	3	2	6
T	3	7	8	18
VPS	0	5	2	7
ST	0	10	9	19
T	1	12	9	22
Physical fitness index*	16 6 ± 1 3	14 9 ± 6 2	9 1 ± 6 4	

* Mean ± standard deviation

exercise tolerance.⁶ Each patient was examined by cardiologists, a psychiatrist, a social worker, and a vocational counselor. Clinical findings, together with results of a standard exercise tolerance test,⁷ were reported at a group conference for a joint formulation of

17 mph on a 10 per cent grade of incline.² This effort is continued for 10 minutes, or to the limits of tolerance if the patient is unable to continue for that duration. This work load increases the oxygen requirement about fourfold.

STANDARD

EXERCISE TOLERANCE TEST

EVALUATION

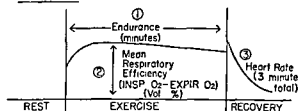


FIG 16-2 Evaluation of over-all cardio-respiratory performance in terms of the Physical Fitness Index of tolerance for standardized exercise

PHYSICAL FITNESS INDEX (PFI)

- Formula: $PFI = \frac{100(\text{Endurance} \times \text{Resp. Effic.})}{\text{Recovery Heart Rate}}$
- Reproducibility: $r = +0.9$
- Normal Range: 13 to 26

The intensity of symptoms was graded and recorded along with duration of effort. Changes in heart rate, blood pressure, efficiency of oxygen extraction from expired air, and precordial lead electrocardiogram were determined for each minute during and for 5 minutes before and after the exertion. Total performance was expressed in terms of the Physical Fitness Index (PFI) (Fig 16-2).⁸ This assumed appropriate motivation to be tested. Performance then varied directly with duration of sustained effort and respiratory efficiency, and inversely with cumulative heart rate for 3 minutes of recovery. The range of PFI for normal subjects is from 13 to 26.

Symptoms and signs exhibited during this

exercise test were subdivided according to the clinical appraisal of functional capacity as indicated in Table 16-3.

1. The incidence of apprehension during the test procedure increased from 6 per cent for Class I to 50 per cent for Class II and 68 per cent for Class III patients. Dyspnea on exertion, the most frequently observed symptom, occurred in 31 per cent of Class I, 67 per cent of Class II, and 88 per cent of Class III patients. Roughly parallel incidences were observed with respect to fatigue. Chest pain was absent in Class I patients, but it was present in 37 per cent of Class II and 53 per cent of Class III patients. Palpitation, dizziness, and leg pain were occasionally noticed by patients in all

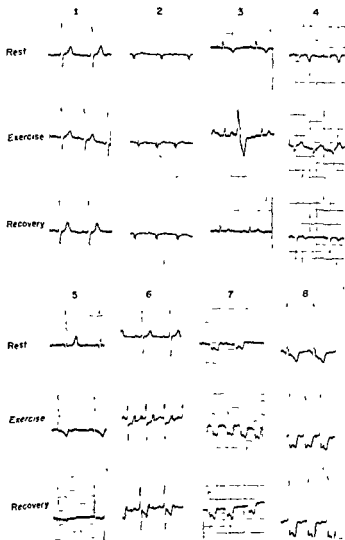


FIG 16-3 Representative precordial lead electrocardiograms recorded on patients with cardiovascular diseases to illustrate types of changes that may be observed during as well as following exercise.

classes. The average duration of time that these persons could exercise diminished from 10 to 8 to 6 minutes for patients in Classes I, II, and III, respectively.

2. Perhaps the most frequent physical sign, other than hyperventilation and tachycardia, was "exertional hypotension." This was defined as either a failure of the systolic pressure to increase or the demonstration of a fall in systolic pressure during exercise. It occurred in a single Class II patient and in nearly a quarter of Class III patients. From other studies it is likely that this is a physical sign of the inability to increase the cardiac output significantly with exertion. Since peripheral resistance decreases with exercise blood pressure falls.*

"Recovery rebound," or an increase in systolic pressure during early recovery above the highest level observed during exercise, occurred in 3 patients in Class III. These abnormal pressor responses are not specific for coronary artery disease since they have been observed in all types of heart disease. Actually, the highest incidence has been found in patients with mitral or aortic stenosis.¹⁰

3. Precordial electrocardiographic changes were analyzed with respect to appearance of ventricular premature systoles, ST displacement of more than 1 mm., and distinct changes in T waves during both exercise and recovery (Fig. 16-3). Class I patients exhibited no irritability and only occasionally some ST displacement or significant change in T wave during exercise. The incidence of T wave changes during recovery was even lower. Patients in Classes II and III showed these abnormalities during exercise, but the incidence of ST displacement was higher during recovery from exercise, especially for individuals in Class III. This difference may be attributed to the fact that the latter patients usually could not walk as long as the others, and did not manifest myocardial ischemia until after the exertion had been discontinued.

4. The range from lowest to highest Physical Fitness Index encompassed both the normal and subnormal zones for patients in

all three classes of functional capacity. Nevertheless, a definite trend was apparent, for the mean PFI was 16.6, 14.9, and 9.1 for patients in Classes I, II, and III, respectively. However, only 70 per cent of the exercise tests were in agreement with the clinical appraisal of functional capacity (Table 16-4). The latter, it should be re-

TABLE 16-4 CLINICAL CORRELATION WITH EXERCISE TOLERANCE AND WORK PRESCRIPTION

Functional capacity	I	II	III	All
Number of patients	16	22	28	26
Relationship to PFT				
Agreement	13	34	20	67
Disagreement	3	18	8	29
Discrepancies due to				
Unsatisfactory history	2	11	6	19
Limited self	1	2	1	4
Medical restrictions	0	0	1	1
Recent convalescence	0	2	0	2
Validity of test				
Due to other disease	0	1	0	1
Due to errors	0	2	0	2
Work prescription				
Unemployable	0	0	4	4
Agency referral	3	17		20
New job	2	4	2	8
Modified job	2	17	12	31
Same job	9	14	2	25

called, was based upon both careful regard for the historical facts and the combined knowledge derived from a multidisciplinary discussion of each patient at a group conference.

5. Discrepancies between functional capacity and exercise tolerance occurred in 29 patients (Table 16-4). On further study, the primary factor was thought to be an unsatisfactory history in 65 per cent of these situations, particularly in those individuals who had difficulty in differentiating cardiac symptoms from emotional reaction to a recent myocardial infarct. Four patients limited themselves unduly, whereas another was excessively restricted by the referring physician. Recent convalescence precluded sufficient activity to permit two patients to experience symptoms. In other words, some patients no longer exerted themselves sufficiently to be aware of potential symptoms, and they were often underestimated with

respect to functional capacity. Indeed, the fact that 86 out of 96 patients had already been restricted by their referring physicians reflected a similar pessimistic tendency.

Although most of the patients who exhibited these discrepancies had not been working for the past 4 to 24 months, virtually all of them had previously had moderately stable work records. Nevertheless, it was of great interest that a psychiatrist considered 20 out of 26 (77 per cent) to have no motivation to work. This was in contrast to a 5 per cent incidence in 67 patients who exhibited satisfactory agreement. Psychiatric evaluation afforded the only basis for predicting which patients were likely to be underestimated with respect to functional evaluation.

The validity of the test was questioned in three patients because of other diseases or errors in the test. One had to discontinue the exertion after 4 minutes because of intermittent claudication rather than symptoms or signs of cardiac impairment. Technical factors in relation to measuring ventilation accounted for false values in two patients.

6 With respect to the work prescription, four patients in Class III were considered unemployable. Three, 17, and 8 persons in Classes I, II, and III respectively, were referred to other agencies for additional guidance and supervision (Table 16-4). Two thirds of all patients were expected to return to work. Whereas most of the employable cardiacs in Class I could return to their same jobs, only two in Class III were considered to have appropriate jobs with energy requirements commensurate with their capacity.

DISCUSSION AND CONCLUSIONS

Evaluation of functional capacity of cardiac patients depends upon a knowledge of symptoms and limitations in relation to activities deemed ordinary by the individual patient. By necessity this involves careful consideration of numerous variables that are largely subjective. Consequently difficulty is

often encountered in defining the intermediate ranges because the criteria are not well defined. Whether or not a particular patient spontaneously seeks relief from continued and customary exertion in order to recover from symptoms may provide a more clearly defined point of discrimination. This criterion seems to be more qualitative than the subtle quantitative distinctions between symptoms with *ordinary* versus *less than ordinary* activity conventionally used to differentiate Class II and Class III categories of functional capacity. Another point of differentiation is the voluntary reduction in working hours by the patient who is symptomatically disabled.

It is of further interest that the clinical appraisal of functional capacity was considered in agreement with the laboratory measurement of exercise tolerance in only seven out of ten patients despite the greater than usual interest and skills of the examining physicians involved in this study. Only the psychiatric discovery of *no motivation* for work suggested which patients were unlikely to be properly evaluated. The majority of patients who exhibited discrepancies were underestimated with respect to functional capacity, for exercise tolerance was usually better than anticipated. This suggests the possibility of exaggeration of symptoms in relation to ordinary activities that may be the result of inappropriate motivation for rehabilitation.

Another advantage of the exercise test, in addition to revealing discrepancies in clinical appraisal of the minority of patients, is the opportunity to reassure the anxious patient. This is done most effectively by the demonstration that he has enough physical capacity to perform the test without harm to himself or alarm to his physician. The implication that the patient has enough capacity to meet many of the energy requirements of ordinary daily activities is usually obvious under these circumstances.

Despite many variables and the subjective nature of such appraisals, satisfactory evaluations of functional capacity can be made in the majority of patients with cardiovas-

cular disorders. Lack of motivation may cause the patient to exaggerate his symptoms and the physician to underestimate the functional capacity. The need for rest and shortening of customary working hours voluntarily by the patient may provide useful additional criteria for differentiating between Classes II and III functional capacity. Finally, additional insight for both patient and physician may be obtained by study of performance during exercise, especially if the procedure is standardized and several variables are recorded.

BIBLIOGRAPHY

- 1 NEW YORK HEART ASSOCIATION, INC. *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Blood Vessels*, by Criteria Committee, HAROLD E. B. PARDEE, Chairman. 5th ed. New York, Am Heart Assn, 1953.
- 2 BRUCE, R. A. Evaluation of functional capacity and exercise tolerance of cardiac patients, *Mod Concepts Cardiovas Dis* 25:321, 1956.
- 3 SPITZER, I. H. The energy requirements of physical work. Translated from a reprint from *REFA Nachrichten* by Purdue Cardiac Project, 1956.
- 4 WUEST, J., GEDDES, D. D., and ENTERLINE, P. Energy expenditure of cardiac workers on the job and a comparison with non-cardiac workers. *Personal communication*, 1956.
- 5 MORRIS, W. H. M. Energy requirements on the farm and in the home. *Personal communication*, 1956.
- 6 SPARKMAN, D. R., and WILSON, J. L. Evaluation of the cardiac worker, *Northwest Med* 55:406, 1956.
- 7 LEVENSON, R. M. Exercise tolerance of the cardiac patient, *Northwest Med* 55:288, 1956.
- 8 BRUCE, R. A., LOVEJOY, F. W., JR., YU, P. N. G., and McDOWELL, M. E. Evaluation and significance of physical fitness for moderate work, *A M A Arch. Indust Hyg* 4:236, 1951.
- 9 BRUCE, R. A., COBB, L. A., KATSURA, S., MORLEDGE, J. H., ANDRUS, W. W., and FULLER, T. J. Exertional hypotension in cardiac patients, *Circulation*. In press.
- 10 LOGAN, G. A., and BRUCE, R. A. Atypical pressor responses to upright posture and exercise in patients with mitral or aortic stenosis, *Amer J Med Sci* 236:168, 1958.

respect to functional capacity. Indeed, the fact that 86 out of 96 patients had already been restricted by their referring physicians reflected a similar pessimistic tendency.

Although most of the patients who exhibited these discrepancies had not been working for the past 4 to 24 months, virtually all of them had previously had moderately stable work records. Nevertheless, it was of great interest that a psychiatrist considered 20 out of 26 (77 per cent) to have no motivation to work. This was in contrast to a 5 per cent incidence in 67 patients who exhibited satisfactory agreement. Psychiatric evaluation afforded the only basis for predicting which patients were likely to be underestimated with respect to functional evaluation.

The validity of the test was questioned in three patients because of other diseases or errors in the test. One had to discontinue the exertion after 4 minutes because of intermittent claudication rather than symptoms or signs of cardiac impairment. Technical factors in relation to measuring ventilation accounted for false values in two patients.

6 With respect to the work prescription, four patients in Class III were considered unemployable. Three, 17, and 8 persons in Classes I, II, and III respectively, were referred to other agencies for additional guidance and supervision (Table 16-4). Two thirds of all patients were expected to return to work. Whereas most of the employable cardiacs in Class I could return to their same jobs, only two in Class III were considered to have appropriate jobs with energy requirements commensurate with their capacity.

DISCUSSION AND CONCLUSIONS

Evaluation of functional capacity of cardiac patients depends upon a knowledge of symptoms and limitations in relation to activities deemed ordinary by the individual patient. By necessity this involves careful consideration of numerous variables that are largely subjective. Consequently difficulty is

often encountered in defining the intermediate ranges because the criteria are not well defined. Whether or not a particular patient spontaneously seeks relief from continued and customary exertion in order to recover from symptoms may provide a more clearly defined point of discrimination. This criterion seems to be more qualitative than the subtle quantitative distinctions between symptoms with *ordinary* versus *less than ordinary* activity conventionally used to differentiate Class II and Class III categories of functional capacity. Another point of differentiation is the voluntary reduction in working hours by the patient who is symptomatically disabled.

It is of further interest that the clinical appraisal of functional capacity was considered in agreement with the laboratory measurement of exercise tolerance in only seven out of ten patients despite the greater than usual interest and skills of the examining physicians involved in this study. Only the psychiatric discovery of *no motivation* for work suggested which patients were unlikely to be properly evaluated. The majority of patients who exhibited discrepancies were underestimated with respect to functional capacity, for exercise tolerance was usually better than anticipated. This suggests the possibility of exaggeration of symptoms in relation to ordinary activities that may be the result of inappropriate motivation for rehabilitation.

Another advantage of the exercise test, in addition to revealing discrepancies in clinical appraisal of the minority of patients, is the opportunity to reassure the anxious patient. This is done most effectively by the demonstration that he has enough physical capacity to perform the test without harm to himself or alarm to his physician. The implication that the patient has enough capacity to meet many of the energy requirements of ordinary daily activities is usually obvious under these circumstances.

Despite many variables and the subjective nature of such appraisals, satisfactory evaluations of functional capacity can be made in the majority of patients with cardiovas-

cardiac disorders. Lack of motivation may cause the patient to exaggerate his symptoms and the physician to underestimate the functional capacity. The need for rest and shortening of customary working hours voluntarily by the patient may provide useful additional criteria for differentiating between Classes II and III functional capacity. Finally, additional insight for both patient and physician may be obtained by study of performance during exercise, especially if the procedure is standardized and several variables are recorded.

BIBLIOGRAPHY

- 1 NEW YORK HEART ASSOCIATION, INC. *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Blood Vessels*, by Criteria Committee, HAROLD E. B. PARDEE, Chairman. 5th ed. New York, Am. Heart Assn., 1953.
- 2 BRUCE, R. A. Evaluation of functional capacity and exercise tolerance of cardiac patients, *Mod Concepts Cardiovas Dis* 25:321, 1956.
- 3 SPITZER, I. H. The energy requirements of physical work. Translated from a reprint from *REF. A Nachrichten* by Purdue Cardiac Project, 1956.
- 4 WUEST, J., GEDDES, D. D., and ENTERLINE, P. Energy expenditure of cardiac workers on the job and a comparison with non-cardiac workers. *Personal communication*, 1956.
- 5 MORRIS, W. H. M. Energy requirements on the farm and in the home. *Personal communication*, 1956.
- 6 SPARKMAN, D. R., and WILSON, J. L. Evaluation of the cardiac worker, *Northwest Med* 55:406, 1956.
- 7 LEVENSON, R. M. Exercise tolerance of the cardiac patient, *Northwest Med* 55:288, 1956.
- 8 BRUCE, R. A., LOVEJOY, F. W., JR., YU, P. N. G., and McDOWELL, M. E. Evaluation and significance of physical fitness for moderate work, *A M A Arch Indust Hyg* 123:6, 1951.
- 9 BRUCE, R. A., COBB, L. A., KATSURA, S., MORLEDGE, J. H., ANDRUS, W. W., and FULLER, T. J. Exertional hypotension in cardiac patients, *Circulation*. In press.
- 10 LOGAN, G. A., and BRUCE, R. A. Atypical pressor responses to upright posture and exercise in patients with mitral or aortic stenosis, *Amer J Med Sci* 236:168, 1958.

CHAPTER 17 INFLUENCE OF HOT AND HUMID ENVIRONMENT UPON THE WORK OF THE HEART*

G. E. Burch

It has been known that a hot and humid environment increases the cardiac output and work of the heart of animals, including man.¹⁻⁸ During experiments concerned with the influence of hot and humid environment upon the normal and diseased cardiovascular system of man, it was noted that acute failure frequently developed in patients with mild or recently compensated congestive heart failure, with acute cardiac asthma and acute pulmonary edema.⁹⁻¹² Indirect observations of cardiac output, consisting of general clinical symptoms and signs (pulse rate, blood pressure, pulse volume, heart sounds, and rheoplethysmographic measurements of the rate of digital blood flow)¹⁰⁻¹³ indicated an increase in cardiac output and work.

More recently, cardiac output and work have been measured by the *direct* Fick

method with cardiac catheterization in normal subjects and in patients with chronic congestive heart failure exposed, at rest in bed, to a comfortable environment (73° F, 60 per cent relative humidity) and then to a hot and humid one (111° F, 86 per cent relative humidity).¹⁴ The hot and humid environment produced an increase in cardiac output and stroke volume (Fig. 17-1). The increase in output was achieved mainly by increase in stroke volume¹⁴ rather than by increase in cardiac rate, as occurs in large part following exercise.

The increase in cardiac output was much greater in the control subjects than in the patients with chronic congestive heart failure. The cardiac output for the control subjects increased from a mean of 5 to a mean of 20 liters per minute and for the subjects with chronic congestive heart failure from a mean of 5 to a mean of 10 liters per minute (Fig. 17-1).

Pressure-volume diagrams were calculated

* Aided by grants from the Public Health Service (H-143) and the Upjohn Company, Kalamazoo, Michigan

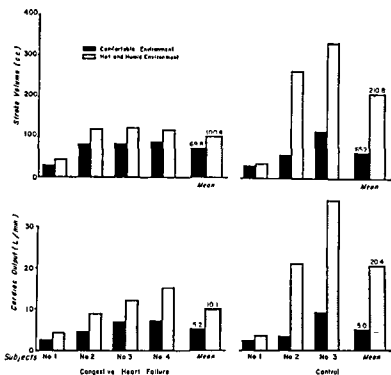


FIG 17-1 Influence of hot and humid environment on cardiac output and stroke volume in 3 control subjects and 4 patients with congestive heart failure (Courtesy *The American Heart Journal*¹⁴)

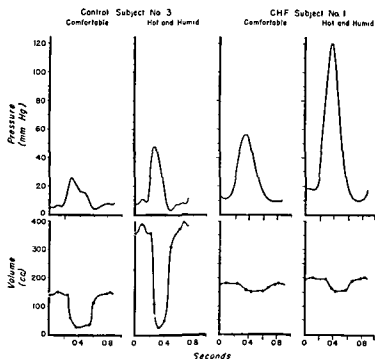


FIG 17-2 Influence of hot and humid environment on the simultaneous time courses of pressure and volume of the right ventricle for a control subject (No 3) and a subject with chronic congestive failure (No 1) (see text for details of assumptions). Considerably higher pressure was developed by the myocardium of the right ventricle of the subject with congestive failure than by that of the control subject, with ejection of much less blood per cardiac stroke, an interesting and not unexpected finding for congestive failure (Courtesy *The American Heart Journal*¹⁴)

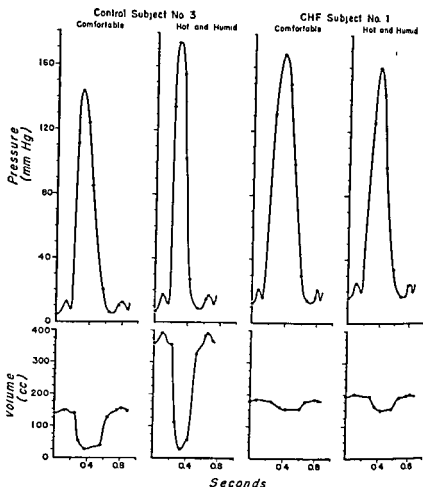


FIG 17-3 Influence of hot and humid environment on simultaneous time courses of pressure and volume of the left ventricle for the same cardiac cycle and same subjects shown in Fig 17-2. Although a proportionately higher pressure was developed by the myocardium of the left ventricle to eject a given volume of blood in the patient with chronic congestive failure than in the normal subject, this difference was not as great as for the right ventricle (consult Fig. 17-2). (Courtesy *The American Heart Journal* ¹⁴)

for the right and left ventricles ¹⁴ The time course curves of pressure in the right ventricle were recorded directly, but those for volume of the right ventricle and for both pressure and volume of the left ventricle were assumed to vary as described by Wiggers ⁵ The pressure curves for the left ventricle were obtained from sphygmomanometric measurements of arterial blood pressure. The end systolic volume used for the control subjects was 25 cc. and for the patients with chronic congestive heart failure it was 150 cc The time course curves of pressure and volume constructed for the right and left ventricles of a representative

normal subject and of a patient with chronic congestive heart failure are shown in Figures 17-2 and 17-3.

The necessary assumptions indicated in the preceding paragraph were reasonable enough to provide pressure-volume diagrams (Figs. 17-4 and 17-5) that appear to be satisfactory and to conform well with physiologic concepts. ^{15,16} The pressure-volume diagrams of the control subjects were increased considerably more in size by the hot and humid environment than were those of the patients with congestive heart failure (Figs 17-4 and 17-5), which indicated that the hearts in failure increased

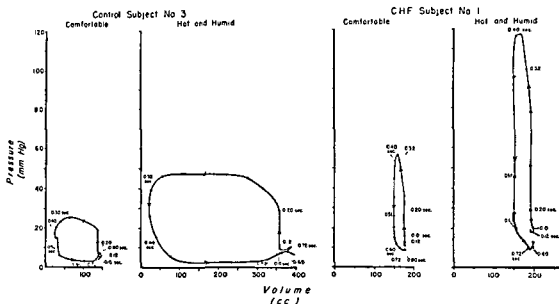


FIG 17-4 Influence of hot and humid environment on volume-pressure-time loops of the right ventricle for a control subject and a subject with chronic congestive heart failure. The more vertical loops for the subject with failure was characteristic for both ventricles (Fig 17-5) of all such subjects studied (Courtesy *The American Heart Journal* ¹⁴)

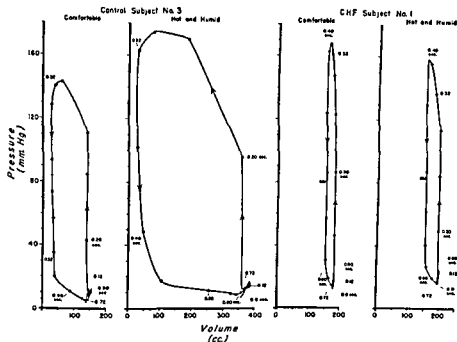


FIG 17-5 Influence of hot and humid environment on volume-pressure-time loops of the left ventricle for the same control subject and subject with chronic congestive heart failure shown in the preceding illustrations. Consult legend of Fig 17-4 (Courtesy *The American Heart Journal* ¹⁴)

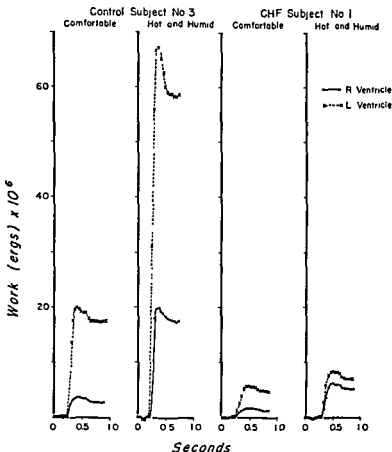


FIG. 17-6 Influence of hot and humid environment on time course of accumulated work for the right and left ventricles for the same cardiac cycle and same subjects shown in the previous illustrations. It is evident that although the subject with failure was stimulated by the humid heat so that the work of his two ventricles was increased, he was not able to do so nearly to the same extent as the normal control subject under the same type of stimulus, an important difference for the decompensated heart. (Courtesy *The American Heart Journal*¹⁰)

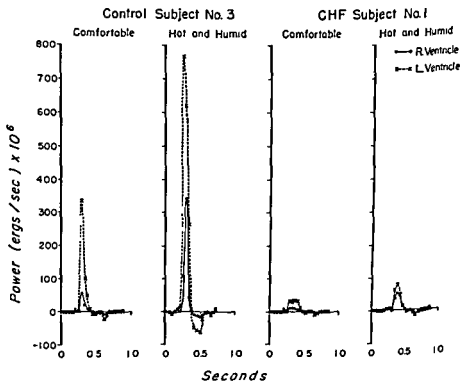


FIG. 17-7 Influence of hot and humid environment on time course of power, or first derivative of curve shown in Fig. 17-6

intraventricular pressure relatively greatly to produce relatively little increase in volume output. On the other hand, the stroke volume output of the normal subjects increased with relatively little rise in pressure. Thus, the pressure-volume diagrams of the subjects with failure were more *vertical* in shape than those of the control subjects

Time-Course Curves of Accumulated Mechanical Work

Mechanical work of the ventricle during a cardiac cycle may best be expressed as

$$W' = \int P dV$$

where P is pressure and V is volume of output. Therefore, by integration, throughout the pulse cycle of the pressure-volume diagrams for the right and left ventricles shown in Figures 17-4 and 17-5, the respective

time-course curves of accumulated work were obtained (Fig. 17-6). These curves not only reveal the differences in the amount of work produced by the ventricles in the comfortable and hot and humid environments but also show the inability of the patients with failure to increase their work output under the stress of a hot and humid environment. This inability of the ventricles to meet extracardiac demands placed upon them may be responsible, in part, for some of the clinical manifestations of congestive heart failure. Furthermore, these curves (Fig. 17-6), as well as the pressure-volume diagrams (Figs 17-4 and 17-5), show not only how futile it is to attempt to increase the work of a ventricle in chronic congestive heart failure but also the inadvisability of allowing such a stimulus to be displayed

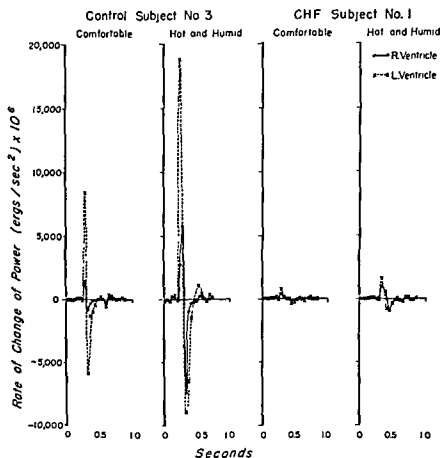


Fig 17-8. Influence of hot and humid environment on time course of rate of change of power, or second derivative of curve shown in Fig 17-6

The first and second derivatives of these work curves are the time course curves of power and rate of change in power (Figs. 17-7 and 17-8). The detailed characteristics of the respective curves may be obtained by careful study of them.

Time-Course Curves of Tension of the Walls of the Ventricles

If it is assumed, for convenience, that the ventricles are spherical, the tension on the wall can be obtained by the equation.

$$T = \frac{RP}{2}$$

where R is the radius of the ventricle and P is the intraventricular pressure. The calculated time-course of tension on the wall, in dynes/cm, is shown in Figure 17-9 for the two ventricles in the comfortable and hot and humid environments considered in Figures 17-2 through 17-8. The ventricular walls of the dilated hearts of the patients with congestive heart failure had greater tension at rest and a relatively greater increase in tension for the work performed than did those of the normal-sized hearts of the control subjects. A study of the illustrations reveals other interesting differences,

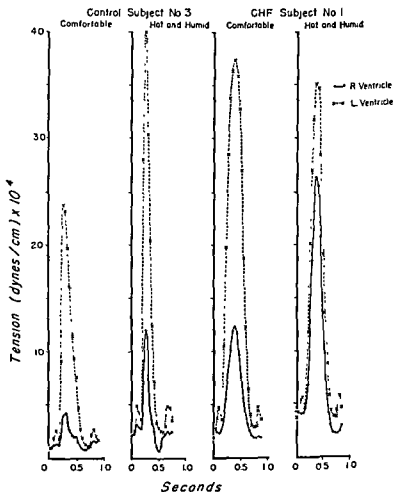
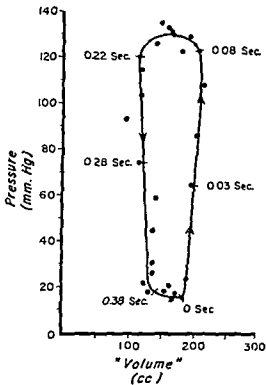


FIG 17-9 Influence of hot and humid environment on time course of tension developed in the walls of the right and left ventricles of the same control subject and subject with chronic congestive heart failure as in the previous illustrations. In the subject with failure, relatively more tension developed to eject less blood per stroke than in the control subject. (Courtesy *The American Heart Journal*)

Fig 17-10 Illustration from an oscillographic recording of the pressure-volume diagram of the left ventricle, during a single cardiac cycle, in a patient with rheumatic aortic valvular stenosis and insufficiency. The points, $\frac{1}{100}$ second apart, are shown as recorded directly by the cathode-ray oscilloscope, whereas the continuous line was drawn manually. The "volume" component is only an approximation of true volume of the left ventricle, as indicated in the text. (Courtesy *The American Heart Journal* ¹⁷)



including aspects of insufficiency of the ventricles of the subjects with congestive heart failure

Automatic Recording of the Pressure-Volume Diagrams

Because of the obvious value of the pressure-volume diagrams in presenting the work of the heart, an attempt was made to record these automatically. Preliminary report of these recordings, with their obvious shortcomings, has been published ¹⁷ By means of direct intraventricular percutaneous puncture of the left ventricle of man through the chest about 2 cm. to the left of the apex beat, and with a pressure transducer and suitable amplifiers, the signal produced vertical deflections of the cathode ray beam of a cathode ray oscilloscope. Variations in "volume," the unsatisfactory parameter, were obtained indirectly by the roentgenologic method of Ring and associates, ¹⁸ with the signal simultaneously producing horizontal deflections of the cathode ray beam of the same oscilloscope. An automatically recorded pressure-"volume" dia-

gram is shown in Figure 17-10. From this diagram, time-course curves of the "work," "power," "rate of change in power," and "tension" in the left ventricular wall were calculated ¹⁷

As stated previously, such recording must be improved, and a program toward this end is now in progress in this laboratory. Many obvious difficulties must be surmounted, particularly to increase the accuracy of the recording of the time-course of changes in volume of the chambers of the heart.

GENERAL DISCUSSION

Measurement of the time-course of work and efficiency of each chamber of the heart as continuous time-course curves throughout the cardiac cycle is necessary. Progress in this direction is being made in attempts to develop satisfactory methods for recording automatically the pressure-volume diagram, as noted in the preceding paragraph and by Rushmer ¹⁹⁻²¹. Time-course curves of efficiency of the chambers are yet to be ob-

tained but will be achieved, at least in the experimental animal. Such records would be of considerable practical clinical importance. Furthermore, since the traces already available are continuous curves, they lend themselves to many obvious and interesting mathematical and electrical analyses and recordings.

The curves presented in this study, even though preliminary and crude, reveal potential and interesting information concerning the time course of the work, power, and tension developed by the ventricles during a single cycle of the heart beat. These curves show the heart to perform relatively more work, with less efficiency, in maintaining adequate blood flow to the tissues, when the subject is in a hot and humid environment. The normal heart was able to increase its work output more efficiently than the heart in chronic congestive failure. The ventricles of the hearts of the patients with failure were able to increase their work under the influence of the hot and humid environment less effectively. Furthermore, these data reveal what a strong stimulus a hot and humid environment can be for cardiac work even while the subject is resting quietly in bed. This type of stimulus should have experimental potentialities.

These data show the importance of making certain that a person with cardiac disease remains in a comfortable environment whenever it is necessary for the heart to rest. The environment, therefore, should be given adequate consideration in the therapeutic regimen for such a patient. Greater use of air-conditioning of the hospital, home, office, and automobile would not only ensure greater cardiac rest but would also improve water and electrolyte balance, as indicated previously.^{11,12}

BIBLIOGRAPHY

- ADOLPH, E. F. *Physiological Regulations*. Lancaster, Jaques Cattell Press, 1943.
- BEST, C. H., and TAYLOR, N. B.: *The Physiological Basis of Medical Practice*. 5th ed. Baltimore, Williams & Wilkins Co., 1950.
- FRANK, J. E. *Howell's Textbook of Physiology*. 17th ed. Philadelphia, W. B. Saunders Co., 1955.
- WRIGHT, S., MAIZELS, M., and JEPSON, J. B. *Applied Physiology*. 9th ed. London, Oxford University Press, 1952.
- WIGGERS, C. J.: *Physiology in Health and Disease*. 5th ed. Philadelphia, Lea & Febiger, 1949.
- BAZETT, H. C.: Physiological responses to heat, *Physiol. Rev.* 7:531, 1927.
- BAZETT, H. C.: The effect of heat on the blood volume and circulation, *J. A. M. A.* 111:1841, 1938.
- BAZETT, H. C., SCOTT, J. C., MAAFIELD, M. E., and BLITHE, M. D.: Effect of baths at different temperatures on oxygen exchange and on the circulation, *Am. J. Physiol.* 119:93, 1937.
- BURGH, G. E.: The normal and abnormal physiologic effects of tropical climate, *New Orleans M. & S. J.* 98:14, 1945.
- BURGH, G. E.: The influence of environmental temperature and relative humidity on the rate of water loss through the skin in congestive heart failure in a subtropical climate, *Am. J. M. Sc.* 211:181, 1946.
- BURGH, G. E.: Influence of variations in atmospheric temperature and humidity on the rates of water and heat loss from the respiratory tract of patients with congestive heart failure living in a subtropical climate, *Am. Heart J.* 32:190, 1946.
- BERENSON, G. S., and BURGH, G. E.: The response of patients with congestive heart failure to a rapid elevation in atmospheric temperature and humidity, *Am. J. M. Sc.* 223:45, 1952.
- BURGH, G. E.: *Digital Plethysmography: Introducing a Method for Recording Simultaneously the Time Course of the Rate of Blood Flow into and out of the Finger Tip*. Modern Medical Monographs No. 11. New York, Grune & Stratton, 1954.
- BURGH, G. E., and HYMAN, A.: Influence of a hot and humid environment upon cardiac output and work in normal man and in patients with chronic congestive heart failure at rest, *Am. Heart J.* 53:665, 1957.
- BURGH, G. E., RAY, C. T., and CROWCHER, J. A.: Certain mechanical peculiarities of the human cardiac pump in normal and diseased states, *Circulation* 5:504, 1952.
- BURGH, G. E.: Theoretic considerations of the time course of pressure developed and

- volume ejected by the normal and dilated left ventricle during systole, *Am Heart J* 50:352, 1955.
7. BURCH, G. E., CROWLIE, J. A., CROFTON, O. and HYMAN, A. Pressure-volume diagrams of the left ventricle of man: a preliminary report, *Am. Heart J* 53:890, 1957
8. RING, G. C., BALABAN, M., and OPPENHEIMER, M. J. Measurement of heart output by electrokymography, *Am J Physiol* 157:343, 1919.
19. RUSHMER, R. F. Pressure circumference relations of left ventricle, *Am J. Physiol.* 186:115, 1955b
20. RUSHMER, R. F., FRANKLIN, D. L., and ELLIS, R. M. Left ventricular dimensions recorded by sonocardiometry, *Circulation Res* 6:684, 1956
21. RUSHMER, R. F. *Cardiac Diagnosis Physiologic Approach* Philadelphia, W. B. Saunders Co., 1955

CHAPTER 18 RELATIVE BURDENS OF EMOTION AND EXERCISE UPON THE CARDIOVASCULAR SYSTEM

Stewart Wolf

The work of the heart can be understood in terms of the demands placed upon it rather than in terms of actual needs for circulation. In fact, adaptations involving the heart, lungs, and peripheral vessels may be of such an order of appropriateness that a moderate exertion may increase relatively little the demands upon the heart. On the other hand, in *untrained* subjects and in situations of anxiety, cardiac hyperfunction may increase the work to an excessive degree.

The circulatory effects that accompany muscular exertion provide extra blood for the working muscles. Heart rate and stroke volume increase while arteriolar dilatation produces a drop in peripheral resistance, thereby preventing an undue rise in blood pressure. Pulmonary ventilation and blood flow are altered and blood flow through the muscles is increased at the expense of visceral flow. The increased heat production incident to muscular effort is mitigated by circulatory changes in the skin that increase heat loss.

Since muscular effort is a familiar stimulus

to cardiovascular function, various workers have devised tests of adequacy of cardiac function and of cardiac reserve. It is well known, however, especially to athletes and trainers, that such cardiovascular adaptations may be made increasingly efficient and economical in terms of cardiac work by practice or training. From the effects of training on the cardiovascular accompaniments of exercise, it would appear that the whole process must be integrated by some regulating mechanism, perhaps in the central nervous system. The likelihood of this is increased by the fact that the circulatory adjustments that are accessible to measurement have been shown to be capable of being produced by meaningful situations such as the contemplation of exercise as well as by the actual undertaking of exercise. Thus it has been shown that stroke volume will increase and peripheral vascular resistance decrease in response to a discussion of exercise, much as salivary and gastric changes are induced by the mere discussion of food.

Such adaptations must inevitably be integrated via the central nervous system, since the impulses that arouse them stem from the interpretive areas of the brain. Indeed, it has been established that a variety of stimuli that owe their force to their peculiar meaning to an individual may evoke cardiovascular patterns concerned with exercise.

Prominent among such factors are life situations that are either consciously or unconsciously threatening to the security of the individual. Situations of pleasurable anticipation may also be associated with tachycardia and an increase in stroke volume. It is as if the bodily changes were occurring in preparation for exertion, e.g., fighting, running away, or active participation in some pleasurable situation. When exercise is actually undertaken under circumstances of stress, the cardiovascular adaptation may be excessive or unduly prolonged during recovery, as if a much greater muscular effort had been anticipated.¹

There are good biological precedents for postulating a bodily reaction as if, or in anticipation of some circumstance. Perhaps the most familiar is the acceleration of salivary and gastric secretion during the pleasurable contemplation of eating. It was not surprising, therefore, that changes identical with those associated with exercise were observed during the mere discussion of muscular effort. Such changes occurred more strikingly during stress without the conscious anticipation of exercise. Similarly, of course, brisk increases in salivary and gastric secretion have been described, not only in the anticipation of eating but during stress unconnected with alimentation.²

EXERCISE TOLERANCE

Changes expected with exercise include temporary tachycardia and increase in stroke volume. The length of the interval before these indicators return to resting levels depends in part upon the amount of exertion undertaken. Tests of exercise tolerance have been difficult to interpret, however, not only because of such factors as training, which

vary the ratio between the amount of exertion and the cardiac work required, but also because factors other than exercise are capable of inducing the cardiovascular adaptations of exercise including tachycardia and increase in stroke volume.³ Thus the work of the heart can be understood in terms of the demand placed upon it rather than in terms of actual need for circulation.

The electrocardiogram as another instrument for measuring exercise tolerance does not provide direct information on cardiac reserve but it may reveal latent evidences of myocardial ischemia brought out by muscular effort. In healthy subjects, changes in the pattern of the electrocardiogram do not ordinarily occur with exercise, when they do, they are thought to indicate a degree of myocardial impairment.

The exercise stimulus used in our studies was the "two-step test" of Master, in which the subject ascends and descends two 9-inch steps for one and a half minutes, the number of trips being adjusted to the age, sex, and weight of the subject.⁴ Most of the tests were done in the morning under basal conditions, but a few were performed at other times of the day, not less than two hours after the last meal. Before beginning observations, the subjects rested on a bed for approximately 20 minutes. The heart rate and the pattern of the electrocardiogram were recorded before and at intervals after the exercise.

THE BALLISTOCARDIOGRAM

The ballistocardiogram recorded with the critically damped, low-frequency instrument of Nickerson reflects the dynamics of heart action as they are modified by the table on which the subject lies and by the weight and contours of the individual studied. Thus, ballistocardiographic records cannot be compared with full satisfaction from person to person. But records taken on the same individual, on the same ballistocardiographic table, and under the same circumstances are comparable. The waves of the ballistocardiogram that reflect force and velocity and volume of blood ejected bear some relationship

to cardiac output, but they have not correlated with sufficient predictability with more generally accepted methods of measuring cardiac output to warrant any quantitative inferences. Using the height and slope of the IJ wave, however, and fitting it into Nickerson's formulas for cardiac output, it is possible to recognize certain dynamic patterns of cardiac function that are seen with frequency and predictability under a given set of circumstances. The method lends itself to prolonged and frequently repeated observation in a single subject and the data thus obtained are satisfactorily reproducible. Alterations in the pattern under stress have been shown to correspond to or combine the characteristics of the changes induced by subcutaneous injection with epinephrine on the one hand (increased cardiac output and peripheral resistance), or with norepinephrine on the other (increased peripheral resistance and reduced cardiac output).

NEUROCIRCULATORY ASTHENIA

Studies were carried out on 35 subjects and reported by Duncan, Stevenson and their colleagues^{5,6}. Of the group, 8 were healthy, asymptomatic persons, 11 had complaints of palpitation and fatigability with or without exertional dyspnea but no demonstrable heart disease. The remaining 16 had hypertensive, arteriosclerotic, rheumatic, or congenital heart disease. The 8 healthy subjects showed comparatively little variation in exercise tolerance. On occasion, however, over a period of 8 months of observation, each of these individuals developed *impaired* exercise tolerance during situations provocative of anxiety or anger.

The 11 subjects who had complaints of palpitation and intolerance for effort would be classified by some as suffering from neurocirculatory asthenia. Essentially, in the absence of structural heart disease, their cardiovascular apparatus overreacted to minor exertion and often displayed an *exercise pattern* when no extra muscular effort was being performed or consciously contemplated.

In many ways these subjects with neuro-

circulatory asthenia were similar from the standpoint of personality adjustment as they were in their characteristic cardiovascular adjustment. They were tense and timid and very sensitive to the hostility of those about them. They lived in an insecure world full of emergencies. For example:

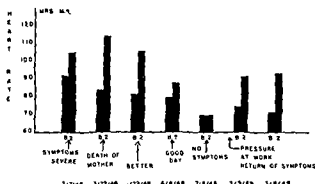
CASE A Impaired exercise tolerance associated with period of anxiety in a young woman with symptoms of neurocirculatory asthenia.

A 24-year-old housewife came to the hospital with the complaint of palpitations that she had had for a year. Other symptoms were tightness in the throat, pain in the left chest, difficulty in breathing, and light-headedness. The symptoms came on more often when the patient was sitting than when she was active. Palpitations were particularly distressing after meals and during any stressful event or disturbing conversation. On Sundays when she was at home and relaxed she was much better.

The patient was the youngest of three children in an immigrant Polish Jewish family. Her father was a butcher. Her mother busied herself in his shop and spent little time with her children. The patient's older brother, 11 years her senior, largely took the place of her father, of whom she saw little. Her brother was a driving, successful young man who, by his example, and to some extent by direct expression, imposed high standards of performance on the patient. When her elder sister married, the patient had to assume a large share of the housework under the direction of her brother. Intensely sensitive to opinions of others, she became excessively preoccupied with neatness and efficiency and disturbed by changes in her routine. At 18 she married a man who was amiable but lacking in initiative and who earned less money than she did. When she was 22, three years after the death of her father of coronary disease, her mother, unable to live harmoniously with her elder daughter, came to live with the patient. The older woman,

her. However, she was deeply dependent on her and prided herself on tolerating her mother when her sister had been unable to do so. In this setting of conflict and anxiety, palpitations began and continued until the patient came to the hospital one year later.

FIG. 18-1. Variations in exercise tolerance from month to month in a patient with symptoms of neurocirculatory asthenia. The heart rate was recorded before (B) and 2 minutes after (2') the standard Master exercise test.



The patient appeared as a neatly dressed, slender woman with an earnest, intense manner. She had rapid heart action (heart rate 90) and warm, moist palms. There was no evidence of structural heart disease. The electrocardiogram was normal as was the basal metabolism (minus 4 per cent). The patient's eyes were prominent and the lids widely opened. She expressed her feelings haltingly and with evident uneasiness about an entirely new experience. Her sensitivity was demonstrated by her statement that even the discussion of someone else's illness evoked palpitations in her. She complained of palpitations when on the subway, chiefly in the morning on the way to work, rarely in the evening after work.

At her first visit, exercise tolerance as estimated from the pulse rate was considerably impaired, as shown in Figure 18-1. As the patient was followed in the clinic, successive tests of exercise tolerance were made. The second test was made a few days after the sudden death of the patient's mother, to which she reacted with considerable guilt and depression. Although the resting heart rate was lower on this day, the exercise tolerance was impaired more than on the previous day. During the interviews that followed, the patient was able to talk more freely about her relations with her parents and brother. She gained some understanding of her emotional development and in addition was reassured concerning the condition of her heart, about which her symptoms had given added anxiety. In the three months following the death of her mother, the patient gradually improved and became free of symptoms. Exercise tolerance was then normal.

The patient remained completely well for another seven months. At this time she arranged for her husband to obtain work at her factory, intending to resign from her own job and have a baby. Her boss, who had obliged her by employ-

ing her husband, hinted that he would discharge him if she left the company. The patient felt frustrated and tense, but was unable either to express her feelings to the boss or leave her job. In this setting she had a return of the former symptoms in milder degree. Although her resting pulse rate was only slightly higher than it had been, exercise tolerance was impaired and continued so for some months thereafter.

CASE B Impaired exercise tolerance associated with periods of anxiety in a tense timid man with symptoms of neurocirculatory asthenia

A second patient with severe symptoms of neurocirculatory asthenia, whose pulse rate reflected marked exercise intolerance but who displayed no evidence of structural heart disease, was a 44-year-old steamfitter's helper who for three years had had repeated episodes of palpitation, fatigability, and weakness. He was the second child of an Irish-Catholic family of seven children. He had been particularly close to his father, a fireman who was accidentally killed when the patient was in his twenties. His mother had died when he was 14. He became a casual, irresponsible person, who was sexually promiscuous and who lacked initiative in his work. He became a steamfitter's helper, but side-stepped all opportunities for promotion in his trade because of an unwillingness to assume further responsibility. In his middle thirties he began to live with a Protestant girl who had already been married twice and who was separated from her second husband. When he was 38 she bore him a child. His palpitations began three years later when his "wife" finally achieved a divorce from her second husband and requested that he go through with his promise to marry her. He felt unequal to the burdens of marriage and con-

tinually put her off. He became sexually impotent and began to note palpitations.

During his early visits in the clinic, his rate was always rapid, usually in the neighborhood of 130. On his first visit his blood pressure was 160/100, but thereafter was recorded in the neighborhood of 120/80. Physical examination, electrocardiograms, and radiologic examination of the heart were entirely normal. The patient was a timid, shy person who avoided the eyes of his interviewer and talked only with effort about his own feelings. Anxiety was always prominent and he was largely preoccupied with gloomy forebodings of the future. He was frightened about the condition of his heart. He became very dependent upon the physician, as he was on his wife, expressing very little hostility directly. The slightest unplanned-for occurrence made him very anxious. During a period of observation in the clinic from December, 1947, to March, 1949, there was a close correlation between his pulse rate at rest and immediately after exercise and his general state of security. The ballistocardiographic pattern corresponded in similar fashion, indicating increased *stroke volume* at times of stress.

When the patient first visited the hospital, he was still unmarried but finally brought himself to accept and welcome the formalization of his relationship with his common-law wife. After this event and the assurance of support at the hospital, he entered a period of relative security and relaxation. Anxiety attacks and palpitations gradually diminished and exercise tolerance improved. A little later he was paired with an uncongenial fellow worker with whom he was unable to exchange hostilities for fear of losing his job. Symptoms of anxiety and palpitations once more increased and concomitantly exercise tolerance became impaired again. Later he obtained new work and improved once more with complete disappearance of symptoms.

Such changes as are observed occasionally in normal subjects and frequently in those with *neurocirculatory asthenia*, characterized by an intolerance to exercise, would not be considered of grave import by most clinicians, and many would attach no clinical significance to them when encountering them in patients without structural heart disease. The same findings, however, observed in a patient with valvular heart disease might have been interpreted as indicating an impairment of

cardiac reserve and might, therefore, have led to an undue restriction of the patient's activities.

STRUCTURAL HEART DISEASE

The remaining 16 patients with various forms of structural heart disease were similarly studied. All these patients complained from time to time of exertional dyspnea and palpitation, which could reasonably be attributed to the structural disturbance in the heart. It was observed, however, that these symptoms occurred at periods of stress and disappeared altogether during periods of relative serenity, although the structural defects of the heart remained unchanged. For example,

CASE C. Young woman with patent ductus arteriosus whose exercise tolerance improved with increased self-confidence.

A 22-year-old girl complained of weakness, dyspnea, palpitations, and dizziness. She had always been warned to "take it easy" since a heart murmur was discovered in childhood. Later, following an unsatisfactory love affair, she became depressed and developed palpitations. She became more and more concerned about her heart. She was found to have a patent ductus arteriosus, which was identified by angiocardiographic examination.

For 10 months she was followed in the clinic, given strong reassurance, and an opportunity to talk over her troubles. As she gained confidence she became more relaxed and her symptoms of effort intolerance disappeared. In Figure 18 2 are shown the comparative exercise tolerance tests during the early period in the clinic, as compared with the findings during the latter part of the period of observation. It is noteworthy that the exercise tolerance was still impaired but to nowhere near the degree that it had been early in the course. Obviously, the congenital deformity in the heart had not changed during this period.

CASE D. Housewife with mitral stenosis whose exercise tolerance improved with reduced anxiety.

A second patient was a 45-year-old housewife who complained of palpitations at rest as well as upon exertion for the previous four years. She was found to have an enlarged heart with mitral

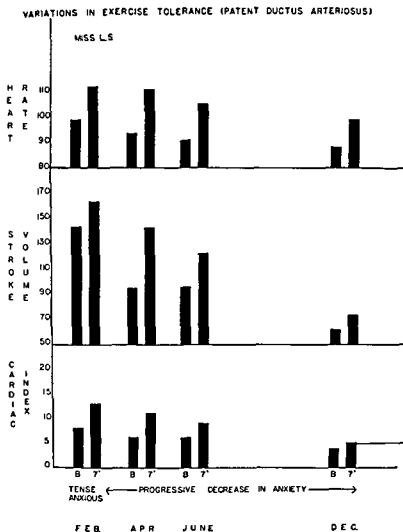


FIG 18-2 Variations in exercise tolerance in a patient with patent ductus arteriosus. Values before (B) and 7 minutes after (7') the standard exercise are given.

stenosis and insufficiency. Frequent extrasystoles were noted. For almost twenty years prior to the onset of her symptoms the patient had wavered in her attachment to two men. One she had loved but, feeling unsure of him, she had married the other one, who appeared to be stronger but proved to be unkind. As her husband increasingly maltreated her she finally resolved to divorce him. Palpitations began during the period of making this decision. As she found difficulty in detaching herself from her husband, she became anxious and tense, blaming both men for her unhappiness.

The exercise tolerance at the height of this period of personal conflict was definitely impaired. Her pulse rate three minutes after the

standard exercise was still over 100, and measurements of the IJ wave of the ballistocardiogram indicated that *stroke volume* was nearly double that of the resting value. Following this, during a seven-month period of observation at the clinic, where she was given considerable emotional support and an opportunity to discuss her conflict, her symptoms greatly improved. A second exercise tolerance test indicated great improvement. The pulse rate three minutes after exercise was slower than the resting rate and the ballistocardiograph indicated less elevation of *stroke volume* than before.

The relevance of stressful situations to serious disturbances in cardiovascular func-

tion has been studied by Reiser, Ferris, and Levine⁷ and recently reviewed. It appears that the added burden of emotional problems on an already marginal cardiovascular compensation may have serious physical consequences for the patient. Any doctor who cares for heart patients must, therefore, concern himself with his patient's problems of life adjustment and must possess or develop the requisite skills to discover, appraise, and deal with them.

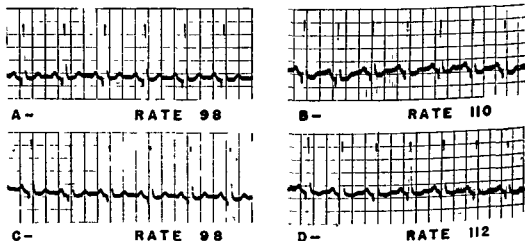
ELECTROCARDIOGRAM

Ordinarily, when there are changes in the pattern of the electrocardiogram during exercise, the assumption is made that there is a disturbance of cardiac nutrition. Duncan and Stevenson⁸ studied 19 patients who displayed changes in ST segments or T waves of a degree considered significant according to the criteria of Master,⁸ following exercise performed during a period of stress. Six of the 19, or approximately one third, failed to show any evidence of heart disease. All six

had the symptoms previously described as characteristic of neurocirculatory asthenia and five of them were under 35 years of age. The other 13 gave evidence of coronary arteriosclerosis. In three patients, one with and two without other evidence of structural heart disease, exercise on a day of relative security and relaxation produced changes in the electrocardiogram that were less impressive than those observed after comparable exercise during anxiety.

In five of the six patients with neurocirculatory asthenia and in all 13 of those with presumed coronary artery disease, it was possible to produce electrocardiographic changes during an interview covering pertinent personal problems and without exercise or conscious anticipation of muscular effort. The interesting fact was that the changes induced during exercise were similar to those occurring during stressful interviews. In 14 of these instances, the heart rate immediately after exercise was nearly identical with that achieved during anxiety and therefore the electrocardiographic complexes were fully

MR. V. C. LEAD II



A - QUIET BEFORE EXERCISE.

B - ONE MINUTE AFTER EXERCISE.

C - SIX MINUTES AFTER EXERCISE.

D - TWO MINUTES LATER. ANXIOUS ABOUT HEARING

FIG. 18-3. Marked depression of T waves in Lead II of electrocardiogram. Essentially the same change was observed after exercise and during discussion of a stressful topic.

comparable. In eight of these the ECG during stress was identical to that following exercise. In four the changes during stress were greater than those induced by exercise and in two instances the changes were qualitatively different.

The changes observed in the electrocardiogram of one subject are shown in Figure 18-3. This patient, 32 years old at the time had symptoms of palpitation and reduced exercise tolerance but gave no evidence of structural heart disease except for the electrocardiogram, which was normal at rest but in which T waves became inverted during exercise or during a stressful interview. Seven years later the patient still showed no further evidence of heart disease.

The mechanisms responsible for these electrocardiographic changes cannot be stated on the basis of the data on hand. They may include coronary ischemia or perhaps merely sympathetic stimulation. The interesting thing is that the changes with stress resulting from threats, symbols, or interpersonal relations are often similar to those following exercise.

The finding is in keeping with the general concept that man during stress may react with his cardiovascular apparatus as if he were about to engage in strenuous muscular activity without any actual awareness of anticipating exercise. Thus the electrocardiogram recorded during severe anxiety in a man with suspected coronary artery disease must be interpreted in the light of these findings. Although the changes may be quickly reversible and may not always be of such grave import as is ordinarily thought, the possibility that repeated or sustained situational stress may lead to irreversible changes must be taken into consideration in planning management.

HYPERTENSION

Cardiovascular changes opposite to those of exercise characterize essential hypertension. Cardiac work is enhanced by the need to overcome an increased peripheral vascular resistance but there is no increase in stroke volume or cardiac output. A similar hemodynamic situation obtains during blood loss.

During rapid withdrawal of blood from donors for transfusion, there was observed an increase in peripheral vascular resistance that balanced the loss of blood volume so that blood pressure levels were maintained. This cardiovascular pattern has also been induced by stressful life situations but with a resultant increase in arterial pressure because no loss of blood volume actually occurred. It is of interest that an increase in blood viscosity as measured *in vitro* accompanies the pressor effects under stress.⁸ Whether or not such factors are pertinent to the pathogenesis of essential hypertension is not known but patients with essential hypertension readily displayed such a hemodynamic reaction under circumstances of suppressed or repressed anger, resentment, or anxiety. From the ballistocardiographic data these instances were marked by an increase in *peripheral resistance* without any increase or often a decrease in calculated *cardiac output*.

CASE E *High resistance type of response to a stressful interview with a restrained, superficially composed hypertensive woman*

A 30-year-old unmarried woman first came to the clinic in 1946 complaining of headaches and easy fatigability. Hypertension had been discovered two years before. Her blood pressure was usually in the neighborhood of 200/110. During the experimental procedure she lay quietly, smiled frequently, and seemed composed, even during a discussion of her relations with her mother, toward whom she felt intense hostility. An underlying truculence was evident in her attitude, however, as manifested by the laconic reply or ironic understatement. During the period of interview her blood pressure rose briskly, a rise associated with a marked increase in blood viscosity and in *peripheral resistance* but none in stroke volume. The course of events is shown graphically in Figure 18-4.

CASE F *High resistance type of response associated with denial of emotional disturbance*

A 30-year-old unmarried secretarial worker was first seen at the hospital in November, 1951. She had been well until 1944 when, following an at-

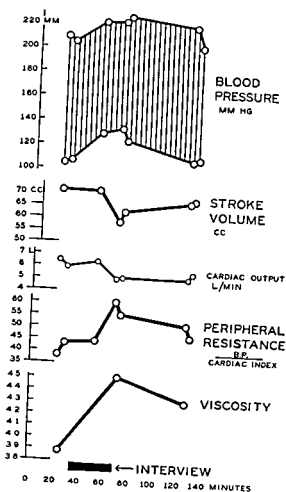


Fig. 18-4 Hemodynamic reaction during stressful interview. Simultaneous measurements showed an increase in blood viscosity associated with pressor response.

tack of acute cholecystitis, a "gangrenous" gall-bladder was removed. Her blood pressure was normal at that time.

She had been the fifth of seven mixed siblings and she was seven years younger than the next older. When she was 7 her mother was found to have diabetes. Two years later she began to require insulin. Because she disliked giving herself injections, her physician suggested that our patient administer the insulin.

Acting on that single injunction the 9-year-old girl faithfully gave her mother insulin twice a day. During the next fourteen years, the only interruption occurred when her mother was in the hospital undergoing a cholecystectomy when the patient was 15. Later, when the patient herself had a cholecystectomy, she demanded to be discharged before her sutures were removed so that she could care for her mother. A few days later at night she heard her mother call. This was not unusual, in fact, for years the patient had been jumping out of bed if her mother was thirsty

or had a cough or was restless. Nevertheless, she jumped up so fast that she tore open her incision. As she entered her mother's room, she found her dying in acute pulmonary edema. Her last words were, "Take care of the family and make sure the youngest boy graduates from college."

Four months after her mother's death, a sudden attack of nausea and vomiting led to the removal of a "gangrenous" appendix. Blood pressure at this time was said to be "180."

Because of her mother's dying request, the patient put aside her plans for marriage. She went to work to earn money enough to send her younger brother through college. Given the nickname "Smiley," at work she was effective, well liked, and highly respected. She always insisted

marry. She cooked for and looked after her father until his death in 1951. Shortly thereafter, and following a massive hemoptysis, she devel-

oped exertional dyspnea, orthopnea, and finally attacks of paroxysmal nocturnal dyspnea. At this time, the blood pressure ranged between 280/170 and 236/138. The pulse rate was 120. The ocular fundi showed Grade I vascular changes; the chest was clear, but the heart was moderately enlarged to percussion and the liver was palpable and tender. The electrocardiogram showed left axis deviation and chest x-ray examination indicated generalized cardiac enlargement. The urine test for protein was 2 plus, blood urea nitrogen 11 mg per cent, urea clearance 63.2 per cent and 62.6 per cent. Intravenous pyelogram was normal.

Following an injection of benzodioxane, there was a sharp rise in her blood pressure and an increase in her symptoms of congestive failure. Tense and exhausted, she was admitted to the hospital. After a brief sleep she was awakened by an intern who insisted upon taking a lengthy history and doing a physical examination, although she had been thoroughly examined in the clinic a few days before. She became intensely angry and spoke out with unaccustomed vituperation. A few days later an experimental interview was undertaken while she was reclining on the ballistocardiograph table. After a suitable control period, the matter of her temper outburst was abruptly introduced. Although she expressed resentment at the treatment she had received, she denied that the episode had upset her in the least. During the day an increase of 40 mm in mean blood pressure was recorded. The pressor response was associated with a fall in calculated stroke volume and with a rise in peripheral resistance.

This latter type of hyperdynamic response would be expected to increase the work of the heart without increasing peripheral blood flow, since blood flow depends more or less directly on cardiac output.

The effects of muscular exercise on the hemodynamics of essential hypertension would appear to be favorable, since there always is produced a temporary reduction in peripheral vascular resistance. Whatever increase in the work of the heart there may be is mitigated by the associated arteriolar dilatation. Despite this it has been customary for doctors to limit the exercise of those with essential hypertension even without cardiac enlargement. There is little evidence to support the

supposition that vigorous exercise has an adverse effect on the cardiovascular apparatus in uncomplicated essential hypertension. In fact, it has been shown among hypertensives as well as healthy subjects that exercise is a reliable way to reduce peripheral vascular resistance. In our own studies of hypertensives we often found arterial pressure at its lowest levels following vigorous muscular effort.¹⁹ Therefore it might seem reasonable to suggest prescription rather than proscription of exercise in essential hypertension in those patients whose hearts are well compensated and not enlarged.

There is relatively little need for concern with regard to *overwork* if the heart is healthy. Although the possibility of bodily damage from overexertion has been entertained from time to time, investigators have failed to produce any very concrete examples of damage to the normal heart from excessive muscular effort. It would appear that motivation provides a sufficient limitation, even in competitive sport, so that general exhaustion stops the effort before undue strain on the heart. We, therefore, concern ourselves with the work of the heart, clinically, only in the presence of a damaged heart.

BLOOD LIPIDS

Currently there is among cardiologists a prominent concern with blood lipids, and especially cholesterol. It is, therefore, attractive to speculate on the possible relevance of blood lipids to the need for fuel in muscular exercise and perhaps throw light on their accumulation in the blood of those subject to coronary artery disease.

Glycogen stores in the liver are not sufficient to replenish and maintain a normal level of blood sugar if glucose were the chief source of energy for heavy physical labor. Neither would a large breakfast and a lunch of two large sandwiches provide sugar enough for the demands of strenuous muscular effort. It is well known that hard-working muscles metabolize fat. Further, it has been shown that the concentration of blood cholesterol and lipoproteins changes very

little following a fat meal or during extreme muscular effort. It is, therefore, evident that a regulatory mechanism exists that tends to maintain a fairly constant blood level of fats while they are being burned for energy, or, in the absence of exertion, while they are being absorbed from the gastrointestinal tract. The work of Siefert and Bader¹¹ suggests that fats may be brought into the blood from depots by a lipid-mobilizing factor secreted in the pituitary gland. Present-day opinion holds that the accumulation of fats in the blood serum may be germane to the pathogenesis of atherosclerosis. It may be that the lipid-mobilizing mechanism is normally brought into play when there is muscular work to be done and, therefore, a need for fats to burn. This mechanism may, like others, be capable of participating in an adaptive reaction *as if*. The hypercholesterolemic patient may be behaving as if he were about to undertake a major muscular effort.

In a recent study of blood cholesterol concentration in relation to emotional stress in patients with coronary artery disease, a very striking correlation was observed between especially high concentrations of cholesterol in the blood and periods of serious emotional conflict.¹² Personality studies indicated that the patients' way of life was Sisyphus-like. That is, they had a special need to do things on their own and often chose the hard way to their objectives, rarely having the opportunity to indulge in restful satisfactions. At the present time the inference is a speculative one, that hypercholesterolemia in some instances may represent a stress reaction with the individual reacting to threatening life situations inappropriately as if great muscular effort were being called for.

Notwithstanding such speculations, it is evident that the work of the heart cannot be estimated with any degree of accuracy with reference to the amount of exertion actually undertaken. If the subject is in training, cardiovascular efficiency will be such that a relatively great load may be carried with a minimum of work. On the other hand, in training or not, the heart may be called upon to work hard with the subject sitting or lying

while contemplating, consciously or unconsciously, some troublesome personal problem.

DISCUSSION AND SUMMARY

Since the heart itself and all the accessory cardiovascular equipment are connected to the interpretive areas of the brain and capable of reacting to symbolic stimuli, it is clear that emotionally stressful situations are as pertinent to determining the work of the heart as are realistic demands for muscular effort. Exercise invariably calls forth the same type of hemodynamic adaptation. It is characterized by acceleration of heart rate, increased stroke volume, reduced peripheral resistance, and a variety of local vasomotor changes in the lungs and elsewhere. With practice or training, the cardiovascular apparatus reacts with increasing efficiency to the burdens of exercise, thus mitigating the work of the heart. Situational stresses may obliterate the effects of training, may exaggerate or prolong the hyperdynamic state of the circulation associated with exercise, or the exercise pattern may be induced by emotional stress without muscular activity. Stress may also induce a variety of other cardiovascular adaptations. Several investigators have pointed out that among these adaptive patterns are two that resemble the effects of epinephrine and norepinephrine respectively.^{10,13,14} The former occurs in situations that are consciously or unconsciously interpreted as threatening. The latter occurs in attitudes. Both include an elevation of arterial pressure, but the work of the heart per unit of blood delivered to the periphery is greater in the norepinephrine-like pattern because of the associated elevation in peripheral resistance.

Experimental correlation of high blood cholesterol concentration with periods of emotional stress has been shown in patients with coronary artery disease. A speculative suggestion has been offered to the effect that great muscular effort may call forth the mobilization of lipids into the blood to sup-

port the energy needs and that under circumstances of emotional stress such a mechanism may also be invoked as if great effort were to be required

Irrespective of the validity of this speculation and its possible relevance to coronary artery disease, it is at least established that increased work for the heart may be required as part of a variety of cardiovascular adaptations to stimuli that owe their force to their particular meaning to the individual

For the healthy heart such extra work is probably of little import. In the presence of cardiac damage or embarrassment, however, the clinician must reckon skillfully with the burdens imposed by emotional strain

BIBLIOGRAPHY

- 1 WOLF, G A, JR, and WOLFF, H G. Studies on the nature of certain symptoms associated with cardiovascular disorders, *Psychosom Med* 8 293, 1946
- 2 WOLF, S, and WOLFF, H G. *Human Gastric Function. An Experimental Study of a Man and His Stomach* 2d ed New York, Oxford University Press, 1947
- 3 HICKAM, J B, CARGILL, W H, and GOLDEN, A. Cardiovascular reactions to emotional stimuli: effect on the cardiac output, arterio-venous oxygen difference, arterial pressure, and peripheral resistance, *J Clin Invest* 27 290, 1948
- 4 MASTER, A M, and OPPENHEIMER, E T. A simple exercise tolerance test for circulatory efficiency with standard tables for normal individuals, *Am J M Sc* 117 233, 1929
- 5 STEVENS, I P, DUNCAN, C H, and RUPLEY, H S. Variations in the electrocardiogram with changes in emotional state, *Geriatrics* 6 164, 1951
- 6 DUNCAN, C H, STEVENSON, I P, and WOLFF, H G. Life situations, emotions, and exercise tolerance, *Psychosom Med* 13 36, 1951.
- 7 REISER, M F, FERRIS, E B, and LEVINE, M. "Cardiovascular Disorders, Heart Disease, and Hypertension," In *Recent Developments in Psychosomatic Medicine*, E D WHITTAKER and R A CLEGHORN (eds) London, Pitman, 1954.
- 8 MASTER, A M, FRIEDMAN, R, and DACK, S. The electrocardiogram after standard exercise as a functional test of the heart, *Am Heart J* 24 777, 1942
- 9 SCHNEIDER, R A. The relation of stress to clotting time, relative viscosity and certain other biophysical alterations of the blood in the normotensive and hypertensive subject, *A Res Nerv & Ment Dis, Proc* (1949) 29 818, 1950
- 10 WOLF, S, CARDON, P V, SHEPARD, E M., and WOLFF, H G. *Life Stress and Essential Hypertension: a Study of Circulatory Adjustments in Man* Baltimore, Williams & Wilkins Co., 1955
- 11 SIEFTER, J, and BADER, D. The occurrence in plasma of an extractable lipid mobilizer (LM), *Proc Soc Exper Biol & Med* 91 42, 1956
- 12 HANMARSTEN, J F, CATHEY, C W, REDMOND, R F, and WOLF, S. Serum cholesterol, diets and stress in patients with coronary artery disease *J Clin Invest* 36 897, 1957
- 13 FUNKENSTEIN, D H, KING, S H, and DROLETTE, M. The direction of anger during a laboratory stress-inducing situation, *Psychosom Med* 16 404, 1954
- 14 MOSES, L, DANIEL, G E, and NICKERSON, J I. Psychogenic factors in essential hypertension, methodology and preliminary report, *Psychosom Med* 18 471, 1956

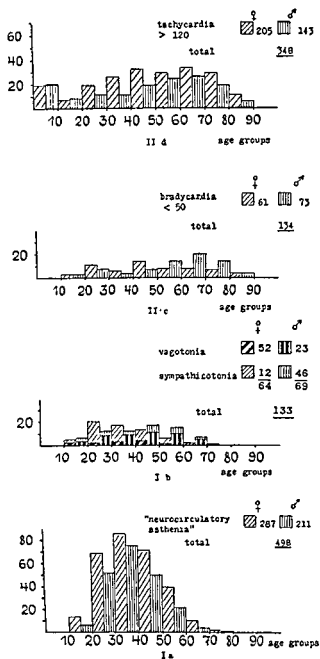


FIG 19-1. Age and sex distribution in patients with functional heart disease, referred for electrocardiograms. II.d Sinus tachycardia II.c Bradycardia I.b Vegetative dystonia, either sympathicotonic or vagotonic. I.a Neurocirculatory asthenia.

induced either by the patient himself, as a protective measure, or by others, not the least of whom are physicians, who misinterpret the condition. The true limit to the working capacity in such cases is not set by physical factors, and cannot be explained in terms of pure hemodynamics. As far as I can see, we need more data on the psychology of work performance. However, I am not sure that everything can be explained on such a basis.

There may still be muscular factors about

which we do not know enough, concerning both the skeletal muscle and the myocardium. I have been thinking somewhat along the lines displayed in Figure 19-4, which is, of course, just a theoretical schematization. I think we know too little about the quantitative chemical composition of muscle. Personally, I have been engaged in quantitative studies on hematin compounds in human cardiac and skeletal muscle, particularly myoglobin and cytochrome C.¹² While we

FIG 19-1. (Contd.). Age and sex distribution in patients with functional heart disease, referred for electrocardiograms III:f Paroxysmal tachycardia III:e Patients with premature beats and a few with the pre-excitation syndrome (From Biorek and Vendsalu*)

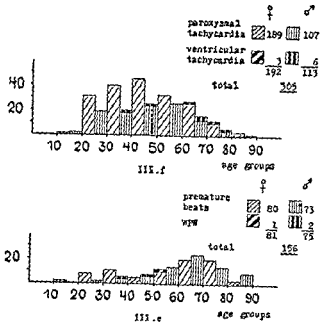
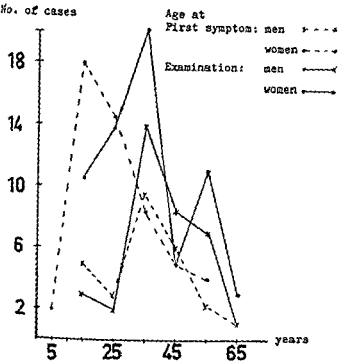


FIG 19-2 Age and sex distribution in 100 private patients with neurocirculatory asthenia. Note that two thirds of the group are women. Furthermore, the first symptoms began at a much earlier age and antedated the examination by many more years in women than men.



studies have not revealed any striking abnormalities of such compounds in the autopsy material at our disposal, the large individual variations in content of these components of the energy-transferring chain are, nevertheless, interesting. One of the sets of data

that is most needed, and one of the greatest gaps in our present knowledge, is that concerning the quantitative chemical composition of muscle in relation to work performance, and the circulatory adjustments for the provision of muscular work.

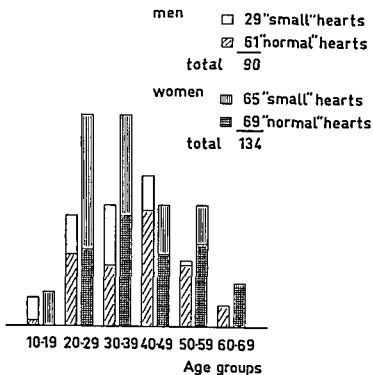


FIG 19-3 Cardiac volumes in males and females in various age groups. *Small* hearts were found in 42 per cent of 224 patients with functional heart disease

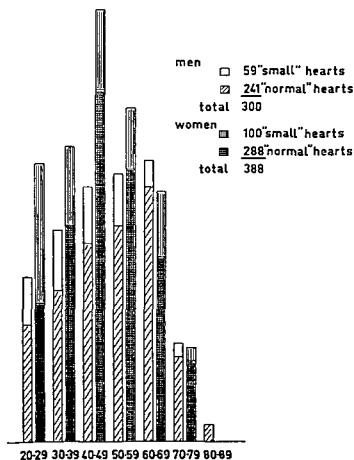


FIG 19-3 (Contd) Cardiac volumes in males and females in various age groups. *Small* hearts were present in only 23 per cent of 688 healthy persons

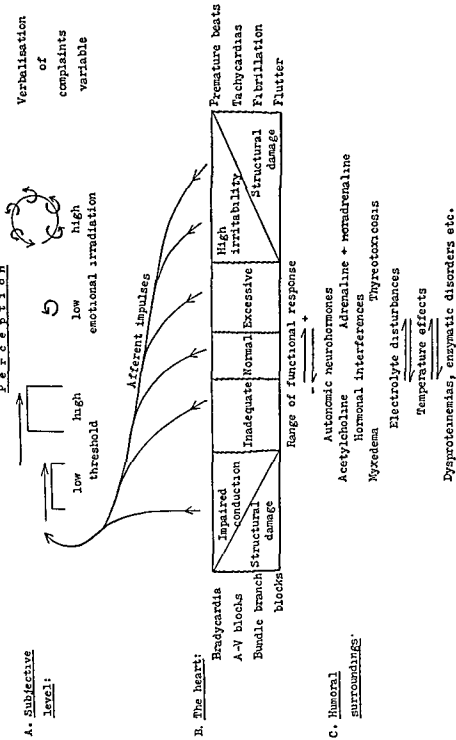


Fig 19-4 Schematic representation of factors which may be operative in the production of functional heart disease and its manifestations (From Borck and Vendsalu *)

BIBLIOGRAPHY

1. WOOD, P. H. : Da Costa's syndrome, *Brit. M. J.* 1.767, 805, 845, 1941
2. WORLD HEALTH ORGANIZATION *Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death*. 6th ed rev International Lists of Diseases and Causes of Death, adopted, 1948. Geneva, World Health Organization, 1948
3. LEWIS, THOMAS *The Soldier's Heart and the Effort Syndrome* New York, Hoeber, 1919
4. SJOSTRAND, T. Das Sporthert, *Deut med Wchn* 80-963, 1955
5. BJORCK, G., and VENDSALU, A.. Studies in functional heart disease a survey of an electrocardiographic material, *Acta med scandinav* 155 361, 1956
6. BJORCK, G. : 100 hjartneuroser, *Scenska lak tidn.* 54 1337, 1957.
7. MASTER, A. M. : The two-step test of myocardial function, *Am. Heart J* 10 495, 1935
8. SJOSTRAND, T., and WAHLUND, H. : Practical view points on hypoxemia test of cardiac function, *Nord. med* 29 599, 1946
9. SJOSTRAND, T. : Exercise tests in heart disease, *Nord med* 50.1353, 1953.
10. FLACK, M. W. : The Milroy lectures on respiratory efficiency in relation to health and disease, *Lancet* 2 637, 693, 1921
11. BJORCK, G., VENDSALU, A. and JOHANSSON, S. : Studies in functional heart disease. the roentgenological heart volume, *Acta Med scandinav* 159.443, 1957.
12. BJORCK, G. : Hematin compounds in mammalian heart and skeletal muscle, *Am Heart J.* 52 624, 1956.

CHAPTER 20 LABORATORY TESTS FOR PHYSICAL FITNESS

Gunnar Björck

In 1956, my fellow countryman, Astrand, reviewed factors affecting human physical fitness, with special reference to sex and age.¹ I would like to quote some statements from his paper

It seems almost certain that if the aim is to examine the physical work capacity of an individual, the examination should be made *during* muscular work. In such a case it is possible to use a submaximal work intensity. Great muscular groups should be engaged in the test work. By this means the oxygen transporting systems can be exposed to a stress without causing local muscular fatigue to be a limiting factor. The work load must be carefully determined and be reproducible. Apparatuses that satisfy reasonable demands are the treadmill and the bicycle ergometer. Investigations should be made during a steady state, i.e., 4–5 minutes or longer after the start of the work, and physiological functions should be measured. The work intensity should not be so high as to make "motivation" play a dominating part. On the other hand, the work level must not be too low. Psychological factors will be able to influence different functions (pulse rate, respiratory rate, etc.) to a great extent when the physical stress is too low.

Tests for physical fitness have been favorite subjects of Swedish physiologists for many years. With the initial stimulation and support of Liljestrand, Nylin has worked with his stair-case test for more than 20 years,^{2,3} later to be followed by Sjostrand,⁴ who employed the bicycle ergometer, and Astrand, who has worked with both the treadmill and the bicycle.⁵ In normal persons, the pulse rate and the oxygen consumption are said to increase in a linear fashion with increasing work. Nylin's test implies the determination of the excess oxygen consumption in relation to resting values after a standard exercise. In his circular stair, Sjostrand, determines the working capacity in kilogrammeter/minute at a heart rate of 170 per minute, in relation to total hemoglobin, as determined by his carbon monoxide method, or to heart volume, and Astrand has studied a variety of factors. The same work loads can, of course, be used to obtain electrocardiographic exercise tests. For particular purposes, other tests have been employed, such as anoxemia tests, orthostatic electrocardiograms, etc.

While normal values have been worked out

by these three methods on healthy people, including students, soldiers, etc., only Nylin's and Sjöstrand's tests have been employed in routine clinical studies. The difficulty inherent in the tests, apart from physiological considerations of the type of work, and the factor to which the measurable change is related, is the considerable overlapping of normal values and values from patients with early or mild pathological changes. The real problem is to construct a dividing line that keeps both *false positives* and *false negatives* within reasonable limits. This is difficult for several reasons; among these is the considerable individual day-to-day variation such as that stressed recently by Sutton and his co-workers.⁶

Furthermore, when one is dealing with persons in whom cardiovascular disease is suspected, the choice of a test and the evaluation of its result must be related to the type of impairment of physical working capacity. I believe there are many different mechanisms to take into consideration. Any test may be interrupted by *discomfort*, either through *pain* in the chest, exhausting *dyspnea*, or a feeling of *dizziness*. The limit of tolerance is set by the *fighting spirit* of the individual. Thus, many factors interplay in tests approaching the *maximal capacity* type. In the submaximal tests, these factors are less important, although still present. In the case of coronary heart disease, pain may be elicited independent of myocardial insufficiency or impairment of the general oxygen transportation system, and pain, in reality, determines the working capacity. Inability of the myocardium proper to respond to a certain external work load in a *normal* way may, furthermore, be due to factors within the muscle fibers or their extracellular surroundings, such as nutritional impairment, structural decay, electrolyte or hormonal disturbances. I believe, however, that such barriers to normal cardiac function as severe valvular stenosis or large shunts must be regarded as separate entities, and we all know that the working capacity of a patient with mitral stenosis may be considerably improved after commissurotomy, a procedure that has nothing to do

with the heart muscle itself. To this may be added the importance of the pulmonary function, which can, perhaps, be disregarded in most persons, but by no means in all. I have seen patients in whom a respiratory neurosis appeared to be the limiting factor for the capacity to work. In his earlier works, Nylin rightly called his test a "cardio-pulmonary function test." Experience with patients belonging to the group labeled neurocirculatory asthenia has brought up the problem whether a deficient regulation of the peripheral circulation, a certain functional inadequacy of cardiac or skeletal muscle or both constitute another set of factors that influence the response to tests for physical fitness, apart from factors of subjective motivation.⁷

The tests usually employed in clinical laboratories are, of necessity, short-term experiments, in which the load is imposed upon the patient rather abruptly. Even if a *steady state* is arrived at, the patient may do much better in his usual surroundings with a proper warming-up and a type of work to which he is accustomed. Figures have been given, which indicate that in manual jobs the worker should have a capacity for oxygen intake at least twice that required for the work per se. While such data are interesting, I have a personal feeling that in our modern society, with its wide spectrum of gainful activities and, even more, in the future society, the purely physiological determinations of working capacity will play a diminishing role in the evaluation of a man's usefulness, and that mental factors will be more important in this respect. There will be no question of assigning people with advanced rheumatic heart disease, or angina pectoris, or emphysema, or neurocirculatory asthenia to physically strenuous jobs, provided they can be utilized in more sedentary occupations. And, if they cannot, our medical advice does not help much, except for a pension certificate. This situation may be different in countries where there is considerable unemployment and social legislation is lacking.

I cannot avoid the feeling that laboratory tests for physical fitness, as regards cardiac

patients, are tools that can corroborate the claims of a patient, or our own clinical impression, of an impaired working capacity in some cases, but are less helpful in predicting a certain range of permissible physical activity

Personally, I grew up in Nylin's laboratory, where his test of functional capacity played a great role in the examination of the cardiac patient. Later, I have felt that such tests serve more as scientific tools than as instruments for the practical management of the individual patient. This is not necessarily the fault of the methods, but is due to the nature of the problem in modern society. Most of our cardiac patients belong to the older age group and have sedentary occupations or are housewives. In these cases we have found the problems of psychological readjustment to be more serious and more neglected than that of physical readjustment. I am, therefore, inclined to believe that one should not try to construct one standard exercise test for all purposes, but rather to choose between different tests for different purposes. Thus, tests for *physical fitness in healthy persons*, athletes, military personnel, etc. might well be *maximal* tests, whereas tests for evaluation of *fitness in cardiac patients returning to manual work* should preferably be fitted to the kind of occupation proposed, and tests of chiefly *scientific interest*, for the study of *circulation during physical stress*, should be adapted to the problem in question. In the social committee of the International Society of Cardiology, we have recently expressed the

need for some kind of international standardization of tests for the functional work capacity of the cardiac patient. I do not believe that it is feasible, nor is it desirable, to claim that one particular test is *the best*. But if, during this conference, we could bring ourselves to an agreement on principles and on the general design of tests for well-defined purposes, I would regard this as a very promising beginning of a new, co-operative project in international cardiology.

BIBLIOGRAPHY

1. ÅSTRAND, P.-O. Human physical fitness with special reference to age and sex, *Physiol. Rev.* 36: 307, 1956.
2. NYLIN, G. Clinical tests of function of the heart, *Acta med. scandinav., supp.* 52, pp. 1-92, 1933.
3. MALMRE, C., NYLIN, G., and SOLLBERGER, A. Normal values of the Nylin heart function test, *Acta Cardiol.* 11: 215, 1956.
4. SJÖSTRAND, T. Volume and distribution of blood and their significance in regulating circulation, *Physiol. Rev.* 33: 202, 1953.
5. ÅSTRAND, P.-O. *Experimental Studies of Physical Working Capacity in Sex and Age*. Copenhagen, Munksgaard, 1952.
6. SUTTON, G. C., SWISHER, W. P., and SUTTON, D. C. Variations in the response of normal persons and cardiac patients to the Nylin heart function test, *Am. Heart J.* 53: 171, 1957.
7. BJÖRCK, G. "Factors Related to Physical Unfitness in Neurocirculatory Asthenia," in *Work and the Heart*, New York, Paul B. Hoeber, Inc., 1959, Chapter 19.

CHAPTER 21 EFFECT OF WORK ON THE HEART

Lucien A. Brouha

The adaptation of the cardiovascular system to muscular work has been extensively investigated. The action of many factors has been studied in animals, frequently by means of radical procedures. In intact organisms and particularly in man, such studies are limited by the techniques that can be used without impairing the subject's health or his performance. This consideration reduces substantially the number of facts that have been observed directly during and after muscular activity.

This presentation is limited to the effect of muscular work on the heart of healthy man. No attempt will be made to explain the mechanisms involved, since these problems are discussed in the *Basic Physiology Panel*. Furthermore, many of these mechanisms and their interrelations have not yet been studied in the intact man. More data are needed and, as long as more information is unavailable, one must realize that some aspects of the over-all picture still remain obscure.

For the sake of clarity, the general effect of muscular work on the heart will be considered first. In a second section the influence

of the major factors influencing these basic reactions will be discussed. A third section will present briefly examples of specific demands on the heart in various occupations.

GENERAL EFFECT OF MUSCULAR WORK ON THE HEART

Heart Rate During Work

The heart rate increases as soon as exercise begins and in many instances even before. This anticipatory rise is commonly attributed to a diminution of the vagal tone reflexly produced by the mental representation of the effort to be made; it originates in the central nervous system. The first increase takes place rapidly and is followed by a second phase during which the level and the behavior of the heart rate are determined mostly by the work load.

Three typical reactions are observed when a constant work load is maintained throughout the exercise period. During light exercise the first increase may be exaggerated and, subsequently, the heart rate diminishes to a

lower level that is maintained as exercise progresses. For a heavier work load the heart rate steadily reaches a rather constant level that persists throughout the exercise. This is described as a "steady state" of heart rate. When the performance is further increased, no steady state can be attained and there is a continued progressive increase in heart rate until exercise stops (Fig. 21-1). In extreme efforts a steady rate can be observed in the latter part of exercise because the heart rate has reached its maximum and cannot increase further. Under these conditions, exercise cannot be maintained for a long period of time and the subject, reaching exhaustion, must stop.

When the work load is variable, the pattern of the heart rate follows each change closely: an increase in load produces an increase in heart rate and vice versa. The relative abruptness of the change depends on the degree of difference in work loads. For a small load increase the heart rate will rise smoothly to establish a new, more elevated plateau; for a marked load increase the change is rapid, the adaptation to the heavier load starts immediately and is achieved in a short time. The reverse is true for decreasing work loads.

For a constant work of sufficient intensity, the duration of exercise also influences the heart rate. If the subject has been able to maintain a steady state for a given time and if the work is prolonged, a secondary and progressive increase in heart rate takes place until exercise stops.

We have been speaking of the typical behavior of the heart rate during exercise involving repeated muscular contractions. In sustained static contractions, such as weight-lifting, the heart rate accelerates slightly, remains unchanged, or even slows down during the effort. The heart rate accelerates when the effort is terminated and it remains accelerated for a time that is determined by the magnitude of the effort and the muscles that were involved.

So far the heart rate reactions have been described when the subject is performing a continuous exercise. When the exercise is not

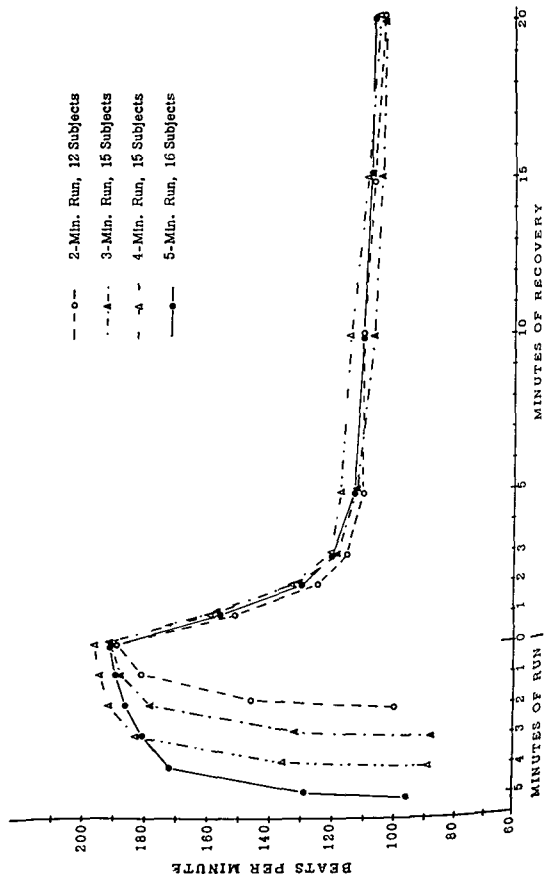
continuous, but repeated several times with periods of rest intervening, the same general reactions are found. For a light exercise the heart rate will increase to the same level during each period of activity. For a harder exercise the heart rate will increase further with each successive period of exercise and no steady state of heart rate can be maintained throughout the total performance in spite of periods of rest taken at regular intervals.

Heart Rate During Recovery from Work

The heart rate decreases almost immediately when work stops. During a few seconds at the beginning of the recovery, the heart rate is very close to the maximum attained at the end of exercise. Then the recovery takes place and the behavior of the heart rate is determined by the preceding exercise.

After light work the heart rate slows down abruptly and soon returns to the pre-exercise resting level. The heavier the load the higher will be the recovery heart rate and the longer it will take to return to the resting level. The duration of exercise also influences the heart rate recovery, provided that the preceding work has been of a sufficient intensity. If the load is light, duration has little influence unless it extends over several hours. For heavier work that can, nevertheless, be maintained in a steady state, the longer the duration of exercise, the longer the recovery to the resting level (e.g., marathon runners, mountain climbers). On the other hand, for comparatively short efforts pushed to exhaustion or nearly so, the duration of the performance does not seem to influence appreciably the recovery processes, and the return to the resting level of heart rate always takes about the same time (Fig. 21-1).

When the exercise is not continuous, the heart rate recovery reactions during the rest periods are determined by the previous work performed and by the duration of the preceding rest periods. For light work the recovery can be complete in a short time even when several periods of exercise have already taken place. This pattern can be described as a *steady state of recovery*. For harder work the recoveries become less and less complete for



a given rest time, so that the recovery reactions remain at a progressively higher heart rate level as the number of exercise periods increases. In a case like this, if a steady state of recovery is to be maintained, the rest periods must become longer as the exercise is repeated. By proper adjustment of the sequence, exercise-rest periods, it is possible to maintain even over a long time a steady state of heart reactions during work as well as during recovery. As soon as the rest time becomes too short, the heart rate increases during successive periods of both work and recovery. A steady state can no longer be maintained and the subject comes closer and closer to his maximum working capacity and his exhaustion level (e.g., tennis players, successive bouts of sprint).

The final recovery to the resting level depends on the total amount of exercise performed and the total amount of rest taken: the more work and the less rest, the longer it takes to recover and vice versa.

For the purpose of comparison between different work loads, various kinds of exercise, or different individuals, the heart rate reactions can be conveniently expressed as cardiac cost. The cardiac cost of work is the total number of heart beats above the pre-exercise resting level that is needed to perform the work. The cardiac cost of recovery is the total number of beats above resting level occurring between the end of exercise and the return to the pre-exercise rate. The total cardiac cost of the performance includes both the cardiac cost of work and the cardiac cost of recovery. In repeated performances of light exercise, the total cardiac cost will remain constant. When heavier exercise is performed, the total cardiac cost will increase from one performance to the next, mainly because the cardiac cost of recovery increases. For heavy exercise, both the work cost and the recovery cost will increase as the performance is repeated. The recovery cost, which can also be called *cardiac debt*, shows greater variations than the cardiac cost and appears

to be an important factor in evaluating the physiological strain produced by muscular work.

Stroke volume and cardiac output are modified by muscular activity. This question, as well as that of heart volume changes during and after exercise, has been thoroughly discussed in a recent review by Asmussen and Nielsen.¹ For the sake of brevity the reader is referred to that article.

FACTORS INFLUENCING THE EFFECT OF WORK ON THE HEART

The response of the heart to muscular work always follows qualitatively the above-described patterns. Nevertheless, it differs quantitatively in the healthy individual according to various factors such as individual fitness, training, age, sex, environment, and acclimatization.

Fitness

For a standard amount of submaximal work, a fit subject will be able to maintain a slower heart rate during work and to recover more quickly after work than a less fit individual. For maximal work the fit and the unfit will reach their maximum heart rate, but the fit subject will be able to perform more work before he reaches that level than the unfit. This is because physical fitness depends to a great extent on the circulatory capacity that is closely related to pulse frequency (Fig. 21-1).

Training

For the same individual performing the same amount of submaximal work, training reduces the heart rate during work and hastens the recovery processes after work (Fig. 21-2). One of the effects of training is to increase the stroke volume so that, in the trained individual, adequate cardiac output is obtained at a lower pulse frequency than before training. Another consequence of this phenomenon is that training will enable one

Fig. 21-1 The similarity of heart rate recovery patterns following exhaustive effort of variable duration.

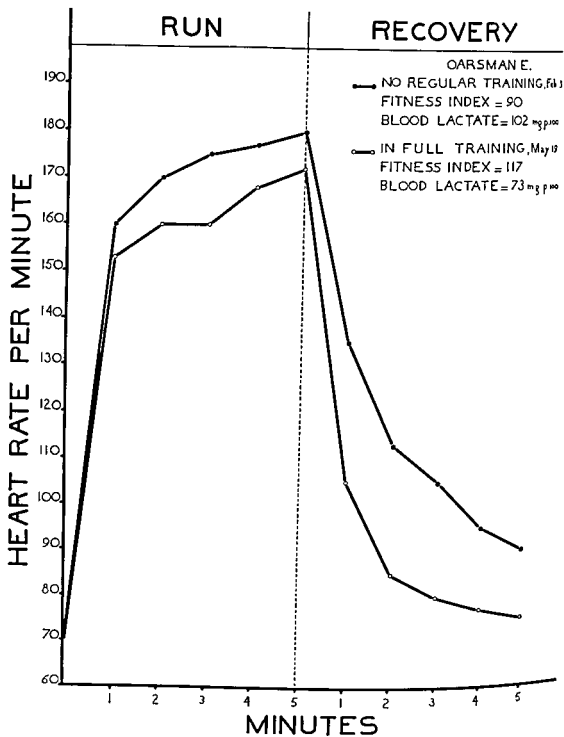


FIG 21-2. Effect of training on the heart rate for a standard amount of exercise and during recovery.

to perform much more work before maximum heart rate and exhaustion are reached

Age

The effect of age has been studied repeatedly in laboratory experiments. They show that after about 25 years of age the maximum heart rate attainable diminishes progressively as well as the ability to perform exhausting work. For submaximal work, older people usually show a faster heart rate than younger ones, but the differences due to individual fitness are considerable and persist until the usual industrial retirement age of 65. Unfortunately, few data are available on the relation of heart reactions and age in various industrial jobs. Figure 21-3 gives an example of increasing average recovery heart rates observed with advancing age in a group of workers performing the same task under similar conditions.

Sex

Heart rate during and after muscular work is quantitatively influenced by sex. In recent experiments, we have seen that for light work (360 kg-m./min.) performed on a bicycle ergometer, women show heart rates higher than

those of the men during work although their recovery patterns are similar. During heavier exercise (540 or 750 kg-m./min.), the heart rates of the women are markedly higher than those of the men and their recovery to the pre-exercise level is slower (Fig. 21-4). In both sexes the maximum increase in heart rate with increasing work follows a straight line. Exhaustion is reached at a lower work load for women than for men (Fig. 21-5).

Environment

Possibly the most important factor involved in determining the effect of work on the heart is the environment in which the work is performed and in which the recovery takes place. Temperature, humidity, and air motion must be taken into account. The effect of the environment on the heart rate is already seen at rest as the environmental conditions become less favorable, the resting heart rate becomes faster. The same is true for the maximum heart rate observed during work, and there is a linear relation between the environmental temperature and the in-

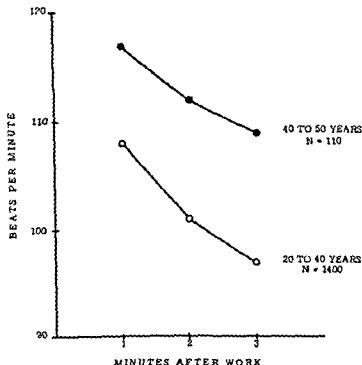


FIG. 21-3 Influence of age on heart rate recovery curves

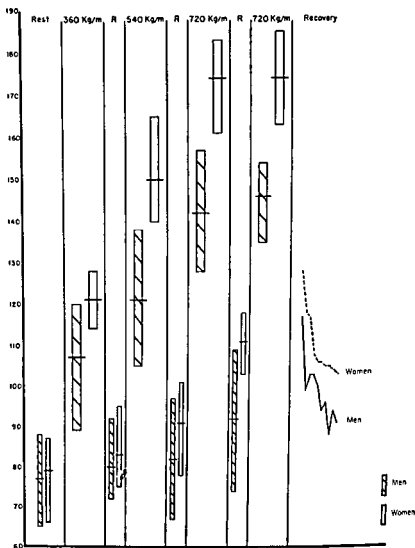


FIG 21-4 Ranges and means of maximum heart rates during work and at 5 min recovery for men and women working at various loads in the same environment

crease of the heart rates both at rest and during work (Fig. 21-6).

Figure 21-7 shows continuous heart rate recordings obtained under three different environmental conditions in which temperature and relative humidity (RH) were varied. Considering that the total work load was the same under the three conditions, the greater cardiac response in the higher temperatures is not due to a different amount of mechanical energy expenditure. As a matter of fact, it is seen (Fig. 21-7) that the oxygen consumption is practically unchanged when shifting from 72° dry bulb (DB) and 42 per cent R.H. (room temperature) to 90°

DB and 85 per cent R.H. (hot wet), and slightly reduced when the conditions are 100° DB and 25 per cent R.H. (hot dry). That is to say that metabolic rate, as measured by oxygen consumption, is not increased by the warm environment, whereas the heart rate is. This acceleration appears to be related

important with increasing body temperature without elevation of body temperature.

The effect of the environment is seen not only during work but also during the recovery. As the environment becomes more severe, the heart rate remains at a higher level for a longer time during the postexercise pe-

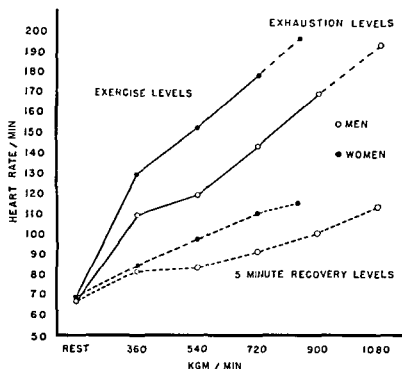


FIG 21-5 Heart rates of men and women at various work levels and during recovery

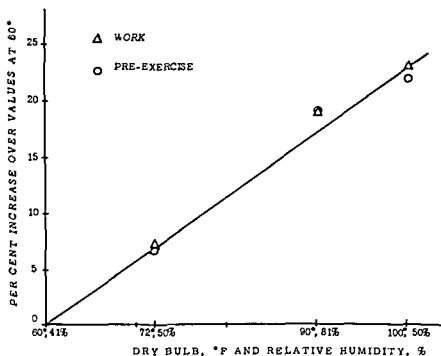


FIG 21-6 Increase of heart rates at rest and at work with increase of temperature.

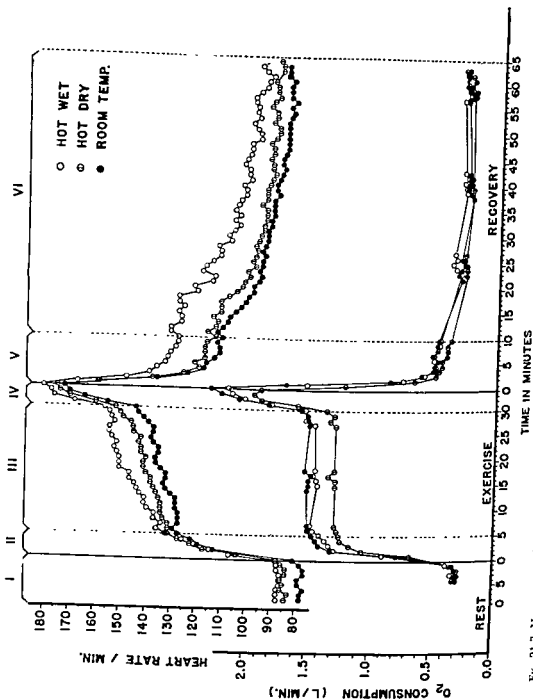


FIG. 21-7. Heart rate and oxygen consumption of male subjects pedalling a bicycle ergometer at 510 kg = m/min during Phases II and III and at 720 kg = m/min during Phase IV, under the different environments.

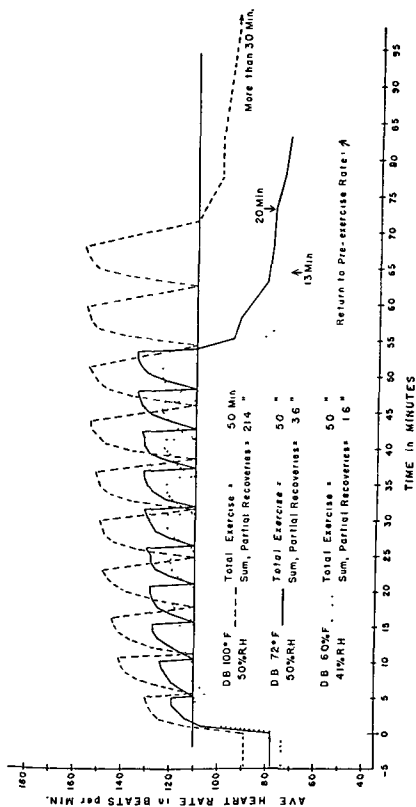


FIG 21-8 Heart rates and recovery times in three environments, for 10 cycles of work and return to 110 beats per minute

riod It should be realized that in warm environments the recovery processes may be unable to restore the resting level of heart rate, since we saw many incomplete recoveries during observations as long as 75 minutes after cessation of work (Fig 21-7, Phases V and VI) In these cases the recovery was complete only after the subjects had moved into more comfortable conditions of temperature and humidity.

The influence of the environment is also clearly demonstrated with successive, intermittent work periods, as in the following experiments

The subjects pedaled a bicycle ergometer at a constant load for 10 successive 5-minute periods Between work periods, sufficient rest was given to allow the heart rate to return to an arbitrarily predetermined level of 110 beats per minute After that rate was recorded, the next work cycle was started within 15 seconds At the end of the tenth ride the subjects were seated in an armchair and their recovery processes studied for 30 minutes These experiments were performed under four different environments; namely,

60° F. and 41 per cent RH, 72° F. and 50 per cent RH, 90° F. and 81 per cent RH, and 100° F. and 50 per cent RH Figure 21-8 shows the average heart rate curves obtained for eight subjects under three of these conditions, the curve at 90° F. and 81 per cent RH. being omitted because it is almost identical with that obtained at 100° F. and 50 per cent RH. Here we see that, as the temperature increases, there is an increase in heart rate and also in the time it takes to return to the rate of 110 beats per minute. The final recovery to the pre-exercise level is complete in 13 minutes at 60° F., in 20 minutes at 72° F., while at 100° F. full recovery is not seen by the end of 30 minutes of rest Figure 21-9 shows markedly greater increases in partial recovery time, that is to the rate of 110 beats per minute, as the work cycles were repeated in a hot environment

In Figure 21-10 the cardiac cost of work and the cardiac cost of recovery to the rate of 110 beats per minute are shown for each cycle under four environmental conditions. These data illustrate the increasing physiologic strain that is observed for the same

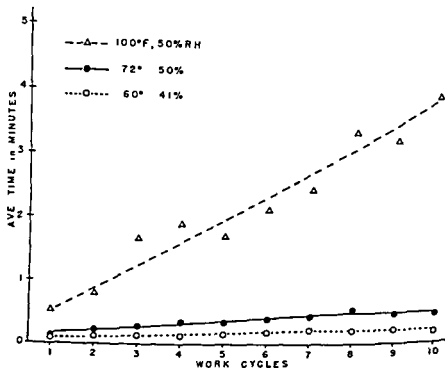


FIG. 21-9. Effect of repeated work cycles on recovery time in three environments.

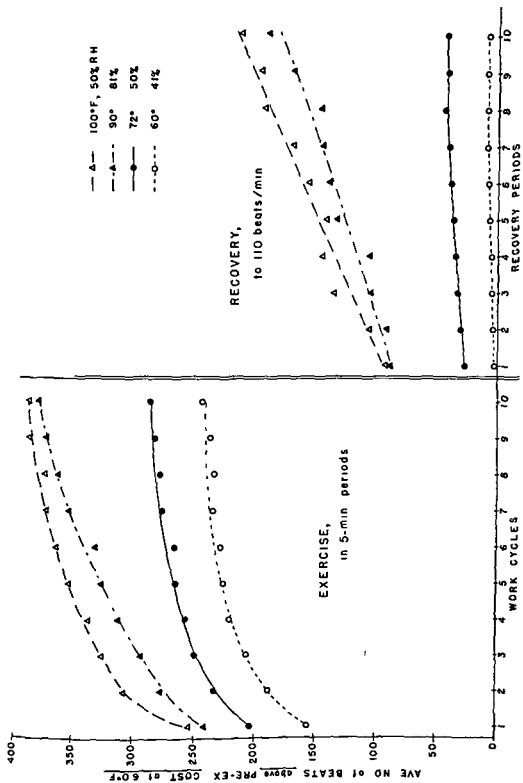


Fig 21-10 Effect of environment on cardiac cost

amount of work performed under progressively more stressing conditions of environment.

Acclimatization

For a standard amount of work performed in warm surroundings, acclimatization reduces the heart rate. Heat-tolerant and acclimatized subjects can work longer and maintain a lower heart rate, mostly because their heat dissipating mechanisms are more efficient.

SPECIFIC DEMANDS ON THE HEART IN VARIOUS OCCUPATIONS

Measuring the cardiac cost of work is impractical in the absence of laboratory facilities. In spite of the fact that telemetering devices have been developed that enable an observer to record continuously the heart rate of a man exercising in the field, this is still an exceptional and costly method. Fortunately for applied research, following the pulse rate variations after the exercise is completed and while the subject is recovering gives interesting and valuable information about the amount of stress he experienced when he was exercising. The principle of this method was developed in the Harvard

Fatigue Laboratory.² It consists of counting the pulse rate by hand at regular intervals during the recovery while the subject is sitting quietly. Usually the pulse is counted from 30 seconds to 1 minute after work stops, from 1½ to 2 minutes, and again from 2½ to 3 minutes. From these pulse counts a *heart rate recovery curve* can be constructed that indicates the actual pulse rates and the speed of recovery toward resting level. As previously indicated, it is seen from these measurements that the heavier the work load the higher the heart rate during recovery and the more slowly it returns to its resting level. In addition, the better the physical capacity of an individual the smaller his increase in heart rate for a standard work load and the more rapid the return to its resting value. Thus, both the physiologic stress and the physical aptitude of an individual for a specific job can be determined from the recovery pulse rates. This kind of measurement is quick and simple as well as inexpensive, requiring only stop watches. It has been used extensively to evaluate the level of cardiovascular stress in industrial operations.³

The onset of fatigue can be predicted from heart rate recovery curves. When no satisfactory recovery can take place between a series

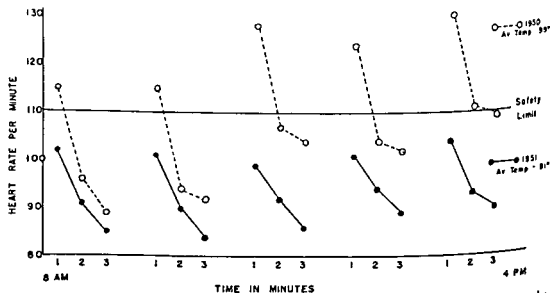


FIG. 21-11 Effect of reducing temperature and humidity on heart rate recovery as work progresses.

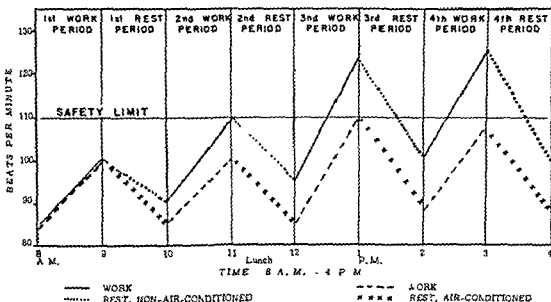


FIG 21-12 Pulse rate changes during an 8-hour shift

of successive operations, the first pulse rate after work becomes progressively higher and the heart rate remains at a high level for a progressively longer time during the recovery period. The return toward the resting level is very slow and definitely points to the existence of *physiological fatigue* in these workers. The upper curves in Figure 21-11 illustrate this situation.

The same method is used to analyze the physiologic effect of various parts of a given job consisting of several operations. Such an analysis indicates which part of a job is the hardest and what should be modified or eliminated in order to reduce the over-all stress. It becomes, therefore, possible to classify jobs according to their physiologic stress.

It is also possible to evaluate the influence of the environment, as shown by the comparison of the upper and the lower curves in Figure 21-11. For the same jobs and the same workers, more favorable conditions of temperature and humidity reduce the demand on the heart resulting in lower heart rates and faster recoveries.

Even if the job is performed under adverse environmental conditions, the over-all strain on the heart can be considerably re-

duced if favorable surroundings are provided during the rest periods, as shown in Figure 21-12.

It has been our experience, over the last 12 years and in many industrial operations, that when the average value of the first recovery pulse is maintained at about 110 beats per minute or below, and when the decrease between the first and the third recovery pulse rates is at least 10 beats per minute, no increasing cardiac strain occurs as the day progresses. Whether this level is produced by the work load alone or by a combination of work load and heat exposure, it appears that such stress can be sustained throughout the shift in a physiologic steady state, provided the sequence of work and rest periods is adequately organized, the recovery between work periods is sufficient, and the heart rate measured at rest at the end of the shift is within normal limits.

In certain cases, aberrant patterns of the heart rate curve can be observed during the recovery. Sometimes the heart rate remains at the same level for the successive pulse readings indicating that no recovery takes place. Sometimes the heart rate increases from the first to the third reading and, when this increase amounts to 10 beats or more,

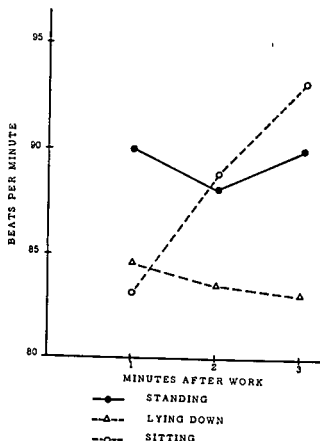


FIG 21-13 Inverse heart rate recovery curve observed in the sitting position.

we classify the phenomenon as an *inverse heart rate recovery curve*. It occurs in the sitting position, as shown in Figure 21-13, and in workers who are involved in moderate physical activity performed in warm and humid surroundings. A typical example is given by employees of cotton mills, where these abnormal curves are frequent. In one of our surveys, as many as 43 per cent of the employees showed inverse heart rate recovery curves with a third pulse reading faster than the first by 10 beats or more. After the third or fourth minute of sitting, this cardiac acceleration stops, but the heart rate remains fast for as much as 12 to 15 minutes before decelerating very slowly.

Similar patterns have been observed in workers exposed to nitroglycerine vapors. It is our belief that they are due to marked peripheral vasodilation with pooling of the blood in the lower extremities while the individual is seated. Because of a poor venous return, the heart must accelerate in order to maintain a sufficient cardiac output.

The preceding presentation and discussion of data lead to the conclusion that the two main factors that determine the heart response to muscular activity are work load and heat load. The consequence is that all jobs involving heavy labor, heat exposure, or a combination of the two are those in which the greatest demand upon the heart is found. Steel mills, aluminum reduction plants, foundries, deep mines, and loading furnaces are among the jobs that are most stressing for the heart. Loading and unloading cars and trucks also produce high cardiac reactions, particularly in the summer time and in warm climates. Often in these cases fast heart rates are observed at the end of the work cycles and the recovery processes become less efficient as the day progresses.³

At a lower level of energy expenditure the heart reactions are determined by the nature of the operation, the speed at which it is performed, the duration of a work cycle, and the environmental conditions.

It is only by studying the physiologic re-

actions produced by a given job that the stress level can be evaluated and that the qualifications of an individual worker can be determined. Unfortunately, many more data are needed and a well-co-ordinated and systematic program should be organized in order to build up a new branch of the scientific study of man: namely, *industrial physiology* or, in an even broader scope, *occupational physiology*. When that is done we will be able to evaluate the human element in any given job and to establish, on a sound basis, the time and sequence of work cycles and rest periods that achieve maximum productive efficiency at minimum physiologic cost. The results will prove, disprove, or correct the basic values of predetermined motion time data frequently used in work measurement. In due course it will be possible to replace the notion of *time motion work cycle* by the concept of *physiologic work cycle*. The latter includes not only effort per unit

of time multiplied by duration of operation but also the physiologic recovery time. This will give a more accurate picture of the effort required by the worker and will furnish quantitative data for estimating fatigue, which remains an essential factor in the utilization of human labor. It will also be possible to establish a sound basis for the selection and the training of the workers so that *the right man for the right job* will no longer be wishful thinking but will really mean *maximum efficiency at minimum human cost*.

BIBLIOGRAPHY

- 1 ASMUSSEN, E., and NIELSEN, M.: Cardiac output during muscular work and its regulation, *Physiol Rev* 35 778, 1955
- 2 JOHNSON, R. E., BROUITA, L., and DARLING, R. C.: Test of physical fitness for strenuous exertion, *Rev canad de biol* 1 491, 1942
- 3 BROUITA, L.: Fatigue measuring and reducing it, *Adv Management* 19 9, 1954

CHAPTER 22 THE FORCE PLATFORM

Lucien A. Brouha

The physiologic considerations discussed so far apply only to work of sufficient intensity and duration to produce reactions that are measurable by the usual physiological techniques. If the work is too short or too light, other methods have to be used. Among them the *force platform*, designed by L. Lauru,¹ has proved very valuable in measuring the efforts involved in performing motions. These measurements are made through sensing elements that are piezo-electric quartz crystals presenting a dielectric polarization when they are compressed. The compression of the quartz crystals is only a few microns and forces from a fraction of an ounce to several tons can be measured. A triangular and very rigid platform permits the location in space of the various quartz crystals so that they can simultaneously pick up force phenomena arising vertically, frontally, and transversely (Fig 22-1). These are amplified and recorded. With the subject on the platform, the whole system is balanced to zero. As soon as the subject moves, the piezo-electric quartz crystals registering in the three dimensions are

submitted to pressure variations. The pressure variations are proportional to the forces applied to the body by the neuromuscular system, and these forces are a measure of the effort required to perform a specific work.

Figure 22-2 shows a record obtained for the three components, vertical, frontal and transversal, when the subject bends the knees, stops and then straightens up to an upright position. It may be seen that bending involves active forces from muscles displacing the body downward and braking forces from the antagonist muscles that control and finally stop the motion. Straightening up also involves an active effort and a braking effort that produce a typical diphasic curve. During any motion, the apparatus continuously and quantitatively records all the dynamic forces and shows all the elements of the motion studied. It is possible to demonstrate by this method of analysis that, of the various motions capable of producing the same final result, some require less expenditure of bodily forces than others. For any mechanical work performed, there is one motion that is the

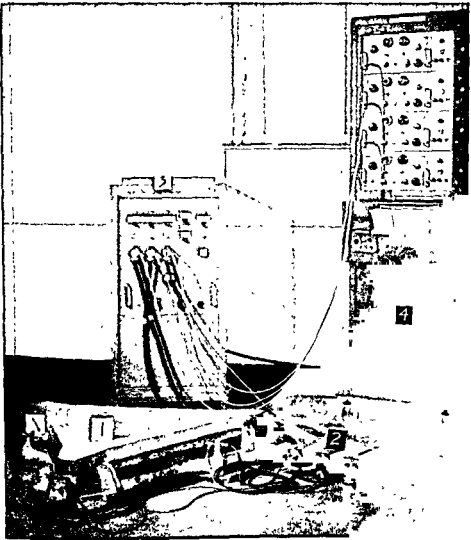


FIG 22-1 1 Triangular force platform The arrow indicates one of the quartz crystals used for measuring forces 2 Electrometers for the vertical, the frontal, and the transversal components 3 Balancing bridge and power supply for the electrometers 4 Sanborn multiple-channel recorder with direct pen writing

most efficient and is physiologically the most economical By adopting these economical motions, jobs can be improved and solutions can be found that in each case represent the best functional conditions The workers can then accomplish their task with a minimum expenditure of physiologic effort, thereby experiencing less strain and fatigue In jobs involving repetitive motions, it is possible to determine the most economic speed It is the one at which the various muscles are func-

tioning at the lowest cost as indicated by the least energy required per unit of motion When a motion involves weight transport, the weight helps the motor forces or the braking forces according to the direction of the motion, and the mechanical work produced is not proportional to the physiologic effort resulting from the muscular contractions A given mechanical work expressed in foot-pounds may correspond to muscular efforts that are quite different according to the

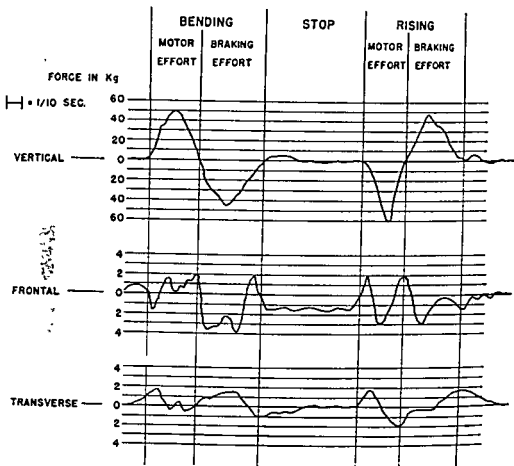


FIG 22-2 Forces involved in bending the knees and straightening up as recorded by the Lauru platform in the vertical, frontal, and transversal components. Note scale change for vertical force.

way of doing the work, according to the weight, and according to the speed of motion that are involved.

Such experiments demonstrate that the external work produced is only one factor in the study of fatigue. By definition they do not take into account the braking forces acting in every motion. These forces, which represent a mechanical loss in the total performance, participate, nevertheless, to the physiologic cost of any muscular activity and are, therefore, a contributing factor to fatigue and to the duration of the recovery period.

When the various motions performed in a job are studied, a complex curve is obtained that is always comparable under similar conditions of performance. The amplitude of the recorded forces is proportional to the corresponding efforts, whether they are producing or braking the motions. They are also

proportional to the energy expenditure as measured by oxygen consumption and to the heart rate during work as well as during recovery from work. The smaller the forces, the lower the oxygen consumption and the heart rate.

place layout. Greater efficiency, smaller physiologic efforts, and shorter time of operation have been achieved by systematic investigation and adequate modifications of certain jobs.

BIBLIOGRAPHY

1. LAURU, L. (Translated by L. A. BROUHA): *Physiological Study of Motions, Adv. Management* 22:17, 1957.

CHAPTER 23 EFFECTS OF VIGOROUS EXERCISE ON THE HEART

Martti J. Karvonen

INTRODUCTION

The normal heart is the heart of a clinically healthy, average citizen, in whom even an autopsy can reveal no cardiac pathology. It is a heart that serves its purpose well. The heart may suffer in many ways and deteriorate as a consequence of pathological changes in the myocardium, valves, coronary arteries, etc. A pathologist can determine whether the heart is structurally normal or abnormal.

The function of the heart is to pump blood. As a pump, the heart may be more or less efficient. The general tendency is to think that a heart changed pathologically is a poor pump, whereas a normal heart a good one. This reasoning may often be valid, but it does not pay attention to an important variable, namely, the changes in the functional capacity of the heart that are produced, on one hand, by training and, on the other hand, by rest.

The best pump is the pump that is most able to increase its performance. Functionally,

the best heart is the heart that can increase its output per minute the most. The ability to raise the cardiac output is critically important for many athletes. When an athlete uses his muscles, they need blood, and with the blood, oxygen and nutrients. In many athletic performances, the functional ability of the heart becomes the limiting factor. Maximum oxygen uptake in liters per minute, i.e., the ability of a subject to take oxygen from inspired air while he performs such effort as running uphill as fast as he can for a few minutes, is correlated with the cardiac output, and often more easily determined than the latter. An essential part of the athlete's training is devoted to building up a large maximum oxygen uptake, although its importance varies from sport to sport.

A clinician may occasionally encounter an athlete with a trained heart that may behave differently in many ways from an average normal heart. Thus, the clinician faces the problem of determining whether the kind of

use that athletes make of their hearts may prove to be deleterious to their health. It is good to have a pump that can pump much blood when needed, but another question is, whether a pump, which has been used frequently to its maximum capacity, will stand the wear of many years as well as a pump handled more steadily and carefully

LONGEVITY OF ATHLETES

Several extensive investigations have been made for determining whether participation in strenuous sports has any effect on the longevity of the athlete. As material for these studies, the student bodies of British and American universities have been used. The choice of sports has been wide, even including sports such as rowing, which is known to place great demands upon the heart. The results of all these studies may be summarized by stating that a student's participation in strenuous sports during his university years has practically no effect upon his longevity. It would seem that strenuous exercise has no important long-term effects upon the heart or other organs.

A study of university students and their athletic achievements may not tell the whole story. The majority of them do not take part in athletics after leaving the university. Quite a different result might be obtained if the athlete were to continue his hobby up to advanced age. There are, indeed, such persons in many places, but rarely as groups, who are easily subjected to a systematic study.

A second criticism of the studies dealing with university athletes is that the groups generally represent a number of different sports that by no means place similar demands upon the athlete's heart. It is reasonable to expect the most marked differences from normal in the subsequent history of those athletes whose hearts present the greatest deviations from the average during the active period.

Skiing in the Scandinavian countries is a sport that makes it possible to overcome the two criticisms presented above. The skiers take part in competitions over several years

In 30 and 50 kilometer races, the mean age of the best competitors has been found to be 30 years, and skiers of even more than 40 years of age have qualified among the best in important competitions. The eligibility for participation in special series for older competitors begins at the age of 35 years. When the interest in competition has passed, skiing does not stop. Long-distance skiing remains a hobby for the old skier, up to the end of his life, whenever possible.

In order to get some idea of the extent to which the oldest living masters of skiing still exert themselves, we sent an inquiry to 61 ex-champions, asking them how many kilometers they had skied during the winters 1955-56 and 1956-57. We obtained 37 answers, from men whose ages ranged from 52 to 83 years. Table 23-1 gives a summary of the re-

TABLE 23-1 RECENT SKIING EXPERIENCE OF EX-CHAMPIONS

Age	Stop- ped at age	Annual skiing now, km			
		1-50	50-100	100-500	>500 Total
40-44	1				1
45-49					0
50-54	3			1	4
55-59	2		1	2	6
60-64	3	2	1	3	9
65-69		3	2	1	6
70-74		1	2	3	8
75-79	2				2
80-84		1			1
Total	11	7	6	10	37

sults. More than two thirds still used the skis every winter. There were three men who skied more than 500 kilometers annually; two of these men were over 70 years of age. The availability of a material such as this provides an excellent opportunity to study the physiological characteristics of old men with an exceptionally good exercise tolerance—if for no other reason, at least for descriptive purposes.

Competitive skiers have a heart that bears several signs of intense training. Some of them will be discussed below. It suffices at

this point only to state that the size of the heart and the electrocardiogram of the skiers differ as much from the normal averages as those of any other group of athletes we have had opportunity to examine.

Skijng races have been arranged in Finland and their results recorded since the latter half of the nineteenth century. We started from a material of 318 skiers whose success in the competitions between 1889 and 1930 had been recorded. Follow-up information could be secured from about half of this material. By applying standard methods of longevity statistics, it was found that the probable age

records of the general male population of

mod has been 66 years, i.e., 7 years less than the life expectancy of the skiers (Fig 23-1) ¹

Such a result may be interpreted in three ways. First, as being due to the primary *potential* of the skiers, which *potential* finds its expression both in success in skijng and in a long life. Such an explanation cannot be refuted easily, but its acceptance would also imply that skijng is the only kind of sport

so far studied, in which such a primary *potential* is of benefit; in other sports success has had no relation to longevity. Second, as demonstrating that skijng lengthens life. This may be so, for the very simple reason that reduction of the functional capacity of the heart by senescence and by chronic disease brings a heart with large functional reserves to the critical limit, at which it no more is able to serve the needs of the body, at a later time than is the case in an untrained individual (Fig 23-2). The third alternative explanation is that skijng is not able to lengthen life except in a population such as that of Finland, where the male life expectancy is considerably shorter than that in the other countries of Western Europe, this difference being due entirely to cardiovascular disease. This line of reasoning would bring the effect of skijng into line with the favorable effect of occupations requiring physical work, as demonstrated in England by Morris and Heady ²

Serum Cholesterol in Skiers

Recent work has indicated that in the development of atherosclerosis, diet is a much more important factor than exercise. We have made a series of determinations of the serum cholesterol of present-day champion skiers, male and female ^{4,5}. The cholesterol values

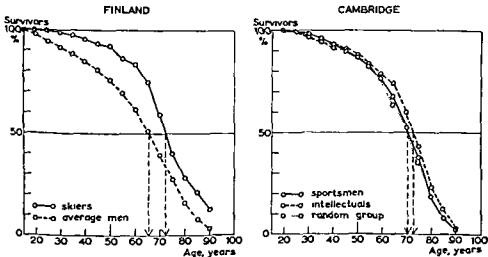


FIG. 23-1 (Left) Percentage survival of Finnish champion skiers as compared with that of the general male population. At the 50 per cent level the difference is approximately 7 years in favour of the skiers. (Right) Corresponding data for the students of Cambridge University, England ¹

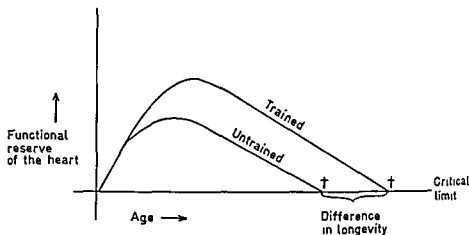


FIG 23-2 Schematic diagram showing the *functional reserve of the heart* as a function of age in trained and untrained men.

were not exceptionally low (Table 23-2). In fact, they were of the same order of magnitude as those found for the Finnish rural population of the same age in the study of Keys and his associates.⁶ Moreover, the participation in a skiing contest did not cause any significant change in the serum cholesterol. Hence, our results do not indicate that an exceptionally heavy form of exercise such as skiing exerts a favourable effect on blood lipids.

TABLE 23-2 SERUM CHOLESTEROL LEVEL OF MALE AND FEMALE CHAMPION SKIERS

	Men	Women
Number of subjects	44	15
Cholesterol mg per cent		
mean	204	194
range	132-307	159-268
standard error of mean	5.4	8.1
Age years		
mean	28.1	24.2
range	21-38	19-29

Whatever the mechanism, certainly the possibility of adding some seven years to the life expectancy merits further study. An expansion and more thorough screening of the material is in progress. Such a result gives quite a new interest to the examination of the characteristics of the skiers' cardiovascular system. Although we might assume, that, in earlier days, training was not as intense as

it is today, the old skiers' hearts and blood vessels in their heyday must have borne some resemblance to those of the champions of today. Further, by making a careful study of present-day skiers we can prepare the ground work for studies of age changes in these athletes in the future.

ROENTGENOLOGIC SIZE OF THE SKIERS' HEART

The heart of a wild rabbit weighs more than that of a domesticated one. The roentgenologic size of an athlete's heart also is larger than that of an average man. However, these two features are not the expression of one and the same phenomenon. Autopsies have been made on endurance athletes with known enlargement of the roentgenologic volume of the heart who have died accidentally; such examinations fail to disclose any hypertrophy of the myocardium. Evidence from roentgenograms taken during the Valsalva maneuver also favors the view that many of the roentgenologically enlarged hearts are not grossly hypertrophic. According to the studies of Sjostrand and his co-workers in Sweden, heart size is very strictly correlated with total blood volume, if the myocardium is not damaged.⁷ The enlargement is essentially a dilatation, and the heart of the trained athlete acts as a blood depot. At rest the amount of residual blood at the end of the systole is increased. When the

athlete starts exercising, more blood is transferred to the periphery, and the heart becomes smaller. This is exactly the opposite of what would be expected from a heart that follows Starling's law strictly. We may say that the heart travels from one extreme of a family of Starling curves to the other. This traveling occurs gradually, not suddenly, after work is begun.^{8,9}

An essential part of the training process of the heart is the development of dilatation at rest, and the ability to change heart size independently of Starling's law. These changes may be secondary to an increase in blood volume. However, it is hardly probable that

TABLE 23-3. HEART SIZE OF CHAMPION SKIERS AND CONTROL GROUPS

Group	Number	cc	Heart volume		Standard error of mean
			cc/24 hr	cc/sq m	
<i>Men</i>					
Olympic training	13	970	46	540	21
National elite	43	904	22	506	12
Army skiers	12	831	46	462	24
Police men (controls)	23	702	27	367	14
<i>Women</i>					
National elite	15	639	20	400	12
Student nurses	14	452	17	270	9

such changes could be achieved solely through increasing the blood volume, without actual training. The heart does not enlarge to the same extent in every form of sport. Figure 23-3 represents mean values for the roentgenologic volume of the heart per square meter body surface from a study that was published in 1948, by a countryman of mine, the late Dr. Soini Pere.¹⁰ The study was comprised of only top-class athletes. The mean values indicate that the largest hearts are seen in sportsmen participating in sports in which endurance is required. Dr. Pere's material did not include top-class skiers. We have measured the size of the heart of a number of male and female skiers of high standard.¹¹ For the best group, 13 competitors in the qualification trials for the Olympic Winter Games at Cortina in 1956, the mean

volume was 970 cc., and as corrected for differences in body size, 540 cc per square meter body surface. When different groups were compared with each other, the athletic standard of the group was reflected in the mean size of the heart (Table 23-3). However, this conclusion could not be applied to individuals within a group of good skiers. We calculated the mean rank of each skier in the most important contests during the winter 1956-57, and plotted it against the volume of his heart (Figs 23-4 and 23-5). If a perfect relationship prevailed, the points should be along a falling line, and the skier with the smallest mean rank, i.e., with most victories, should have the largest heart. However, the points appear scattered without any regularity. The conclusion is to be drawn that, although enlargement of the heart at rest is a result of endurance training, its extent is, by far, not the sole determining factor of athletic success.

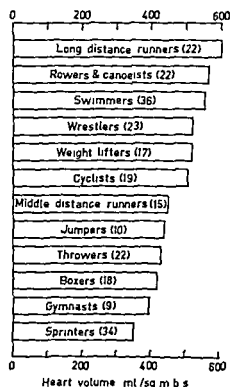


FIG 23-3 Heart volume in millilitres per square meter body surface of top class Finnish athletes (Recalculated from Pere's data¹⁰)

In animal experiments, training does produce an increase in the weight of the heart, but, even here, hypertrophy is not in direct relation to the improvement of the animal's exercise tolerance. Hakkila¹² trained guinea pigs by running, and the animals that ran best did not develop any more hypertrophy of the heart than the poorer runners.

ELECTROCARDIOGRAM OF THE SKIERS' HEART

The electrocardiogram of the individual in athletic training shows typical characteristics that differ from those of the heart of the average normal person. At rest, extremes of bradycardia may be seen; pulse rates below 50 per minute are quite usual, and pulse rates as low as 35 per minute are encountered. Bradycardia is often associated with slow conduction. Table 23-4 shows the con-

TABLE 23-4 CONDUCTION TIMES IN THE RESTING ELECTROCARDIOGRAMS OF 21 CHAMPION SKIERS¹⁴

PQ		QRS		QT (Per cent of normal mean)	
Sec	n	Sec	n	Per cent	n
0 20	9	0 10	16	112	9
0 20	6	0 10-0 12	4	112-118	5
0 21	1	0 12	1	118-124	4
0 22	1	Total	21	124-130	2
0 23	2			130	1
0 24	1			Total	21
0 25	1				
Total	21				

duction times in a group of 21 participants in a qualification meet for the 1956 Winter Olympic Games, studied by Rautaharju in our laboratory.¹³ When looking at the electrocardiograms, the most striking characteristic is the *large voltages*. Figure 23-6 shows

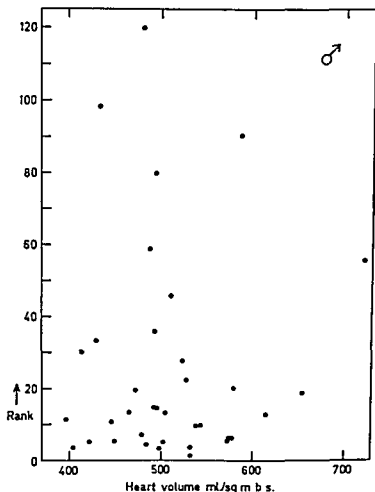


FIG. 23-4 Mean rank of Finnish male champion skiers in the most important competitions of 1957, as plotted against their heart volume. If the heart volume determined the success in skiing, a negative correlation would be expected.

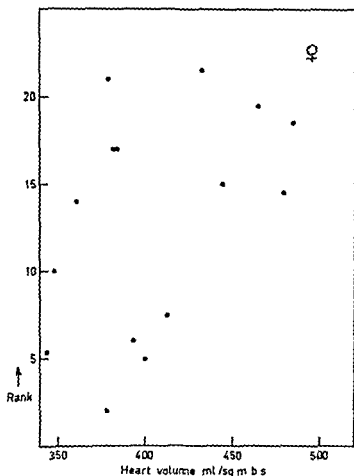


FIG. 23-5 Mean rank of Finnish female champion skiers in the most important competitions of 1957, as plotted against their heart volume. Cf Fig 23-4

the voltages of the R, S, and T waves as recorded from the precordial leads at the level of the fifth intercostal space at the sternum. As a comparison the mean values of a normal control group of the same age structure are used, representing various types of occupation. The R waves of the skiers are almost twice as large as those of the controls in the left precordial leads, whereas there is no difference over the right precordium

Nevertheless, in the left precordial leads, the ventricular activation times were quite normal. The S waves are about the same in both groups. In the left precordial leads, the T waves of the skiers are about twice as large as those of the controls.

The electrocardiograms were also analyzed according to the spatial vector method of Simonson¹⁴. Table 23-5 presents a comparison of the QRS and T vectors of the skiers

TABLE 23-5 MEAN VALUES OF SPATIAL QRS AND T VECTORS OF 21 FINNISH CHAMPION SKIERS AND OF A CONTROL GROUP. P = LEVEL SIGNIFICANCE OF THE DIFFERENCE

Group	n	QRS vector			T vector			
		H°	I°	Mag	H°	I°	Mag	A°
Skiers	21	-1.6	33.1	15.8	32.4	51.2	5.4	31.0
Controls	33	-29.7	35.9	10.3	51.1	60.2	3.8	60.4
P		<0.001	>0.05	<0.001	<0.001	>0.05	<0.001	<0.001

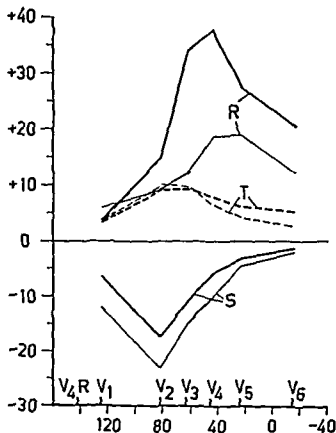


FIG 23-6 Mean amplitudes (0.1 mV) of R, S, and T waves in the precordial leads of champion skiers (thick lines) and of an age matched control group (thin lines).

with those of the controls. There is a shift of the horizontal angle of QRS (H°) to the right, the difference from the controls is statistically significant. In the vertical angle (V°), however, there is no difference between the skiers and the control group. The magnitude of the QRS vector (Mag) of the skiers is about 50 per cent greater than that of the controls. The T vector of the skiers points to the left of that found in the controls (H°). Again, there is no difference in the vertical angle (V°), but the magnitudes (Mag) are different: the T vector of the skiers is 40 per cent greater than that of the controls. It is of interest to note the small angle (A°) between the QRS and T vectors in the skiers as compared with that in the controls. In myocardial ischemia, this angle generally increases. The deflections of large amplitudes sometimes seen in pathological hearts, thus show distinct differences from those encountered in the electrocardiogram of the trained athlete.

The magnitude of QRS and its vertical angle show a distinct positive correlation in

the skiers (Fig 23-7). A similar tendency could be observed also in the small Finnish control series. However, in the normal material studied in Minneapolis by Simonson and Keys there was no such correlation.¹³ A larger material will be required to show

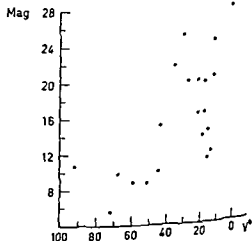


FIG 23-7. Magnitude of the QRS vector in champion skiers' electrocardiograms plotted against the vertical angle.

whether there is a real population difference in this respect.

The electrocardiographic picture of incomplete right bundle branch block is common in the heart of the trained athlete, it occurred in five subjects of Rautaharju's series.¹³ Even complete right bundle branch block occurs; there was one example among the 21 individuals in this group. It is questionable whether these electrocardiographic changes, particularly the large voltages, may develop only as signs of functional adaptation. There are many factors that can cause increased voltages. Hypertrophy of the myocardial fibrils is one of them.

THE TRAINING PROCESS

Vigorous exercise evidently can produce profound long-term changes in the function of the heart. The mortality experience of ex-skiers makes these changes appear harmless, possibly even advantageous. All physical work and exercise does not lead to equally marked changes in the heart. What are the specific factors in the training of skiers and of other sportsmen that lead to the cardiac changes in the athlete?

We made pilot experiments in order to clarify this problem. The subjects, previously untrained medical students, ran on a treadmill half an hour daily for four weeks. The speed of the treadmill was adjusted according to the pulse rate of the subjects, so that each subject ran at a specific predetermined pulse rate. In some of the training experiments, the exercise tolerance improved gradually, and the pulse rate during work tended to become lower than it was originally. Then, the speed of the treadmill was increased, to keep the heart rate at the original level. An effect of training on the working heart rate was thus demonstrated by an increase in the running speed.

A typical example of the type of results is the following. In one series, in which the subject was running at a heart rate of 135 per minute, there was no change in the working heart rate, whereas the same subject manifested a progressive improvement,

when he was running at a high heart rate, at 150 per minute. It would appear that in order to improve the exercise tolerance of the heart, the intensity of the training has to be above a rather high threshold value. The intensity of training may be expressed also as a percentage of the training pulse from the total range of heart rates from rest to the maximum attainable by running. Sixty per cent of the range appears to be the critical rate, above this level the training is effective, below it the training is ineffective (Fig. 23-8).

A decrease of the working pulse rate was by no means the only cardiovascular manifestation of training. Even in the absence of changes in the working pulse rate, training resulted in (a) a slowing of the resting pulse rate, (b) a more rapid recovery after standard exercise, (c) a decrease of the maximum pulse rate attainable by running; and (d) a marked improvement in the subjective exercise tolerance. The latter manifestations may be due to changes in the peripheral circulation rather than in the heart itself. It is possible that the peripheral circulation may be trained without causing training effects in the heart.

The necessity of raising the pulse rate to high values in order to train the heart makes it obvious that, in the majority of the present-day occupations, the heart is seldom effectively trained. A pulse rate of 125 or 130 per minute is considered the maximum to be permitted in continuous work.¹⁴ In actual industrial conditions even a pulse of 120 per minute is relatively rare, the majority of workers engaged in physical labour may have pulse rates of approximately 100 per minute. On the other hand, in less well controlled occupations such as farming and lumbering, spells of harder exercise are often interposed, with the possibility of producing a training effect on the heart. In industrial occupations the work remains a secondary determinant of the exercise tolerance. If an office worker takes part in some vigorous recreational activity such as tennis, the exercise tolerance of his heart determined by a step test is distinctly superior to that of a

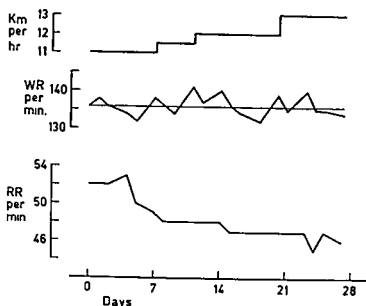


FIG. 23-8 A training experiment in which a tendency of the working heart rate (WR) to decrease necessitated an increase of the running speed (km per hr). The resting heart rate (RR) also decreased during the training period.

man working eight hours daily at a fairly heavy, monotonous job.

The demonstration of critical *threshold* levels in training shows that in attempting to study, and to interpret, the effects of exercise upon the heart, upon cardiovascular morbidity and upon longevity, pooling all kinds of exercise under one heading may produce a crucial dilution of different types of effects. Where the exercise can be defined as accurately as that of a postman or of a conductor of a double decker bus in London, no ambiguity ensues. However, pooling all kinds of sportsmen into a single group may lead to a rather heterogeneous entity. In future studies, a clear-cut definition of the amount and type of activity appears desirable. Then the characterization of the changes produced by activity rests on a firmer basis.

HEART AND PERIPHERAL CIRCULATION

Schematically, we can divide the carrying of oxygen from the lungs to the active muscle fibers into three functions. The first function is the pumping action of the heart; the adaptation of the heart to exercise is obviously measured by its ability to increase its output per minute and per beat. The second function is the transfer of oxygen from the blood to the tissues. A large arteriovenous oxygen gradient in the muscle means an in-

creased contribution of this effect to cover the needs of the tissues for oxygen. A large arteriovenous difference obviously reduces the need for an increase in the cardiac output. On the other hand, a large arteriovenous oxygen gradient in the working muscle lowers the oxygen tension at which the muscle works, and brings the muscle nearer to anaerobic conditions. The ability of the muscles to work at a low oxygen tension is, therefore, the third function determining the adaptation of the organism to exercise. The adaptation to low oxygen tensions may be of two kinds: (a) a lowering of the critical oxygen tension at which aerobic processes become impossible, and (b) an ability to stand higher rates of production of anaerobic metabolites, notably lactic acid. The latter signifies, on one hand, an ability of the central nervous system to work at a high lactic acid concentration, and, consequently, at a low pH of the blood. On the other hand, the organism may improve its ability to withstand high rates of lactic acid production by increasing the rate of its simultaneous elimination, which is largely a function of the liver.

An endurance athlete is known to be able to increase the first function, his cardiac output, more than the average man. The contributions of the second and third functions to endurance are less clear. Asmussen and

Nielsen¹⁷ have pointed out that the arteriovenous oxygen difference in the working muscles shows no tendency to increase with the intensity of exercise in studies in which the subjects have been well trained,^{18,19} whereas in studies with average subjects the arteriovenous oxygen difference in the muscle increases with oxygen uptake.^{20,21} These results would suggest that, in the trained organism, the arteriovenous oxygen gradient in working muscle will remain rather steady, the increase of the cardiac output being the dominant means of adaptation to exercise (Fig. 23-9).

A quite contrary conclusion was reached recently by Mellerowicz, who made a study of a number of top-class endurance athletes in West Berlin.²² The circulation times at rest were very low, with the sodium succinate method of Greenfield²³ the mean time for the arm vein to swallowing reflex in sportsmen was 27.3 seconds, while the mean for average men was 14.8 seconds. Furthermore, a calculation of the cardiac output from pulse rate, blood pressure, and the velocity of the pulse wave at rest gave remarkably low values. Although little reliance can be given to the results of this very indirect method for determining the cardiac output, the results suggest, nevertheless, that top level endurance training also implies an adaptation of the periphery. The heart of a trained athlete at rest does considerably less work than that of an average person. By taking the trouble to train his heart and

circulation, the endurance athlete not only gets the benefit of having larger reserves, but also may gain by shifting the balance between the heart and the peripheral circulation in such a way that the heart gets an easier share and the periphery does its job in a better way.

PHYSIOLOGY OF SPORT AND CLINICAL CARDIOLOGY

The clinician is more interested in a patient than in an athlete. He may ask whether experiences gained in endurance training have any application to the problems of a cardiac patient. This is as yet an unwritten chapter. We should like to know to what extent and how an individual cardiac patient can train his heart without the risk of untoward effects, or whether, and when, systematic rest is to be preferred to training. We should like to know, also, whether a cardiac patient can train his periphery and thus relieve the burden upon his heart. It is in problems such as these that physiological and medical studies on sportsmen may be of value to clinical cardiology.

BIBLIOGRAPHY

- 1 KARIÖNEN, M. J., KIHILBERG, J., MAATTA, J., and VIRKAJARVI, J. Longevity of champion skiers, *Duodecim* 72:893, 1956 (In Finnish)
- 2 ROOK, A. An investigation into the longevity of Cambridge sportsmen, *Brit M J* 1:773, 1954

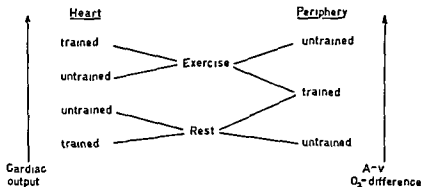


FIG. 23-9 A hypothetical diagram of changes in the cardiac output and the arteriovenous O₂ difference with training

3. MORRIS, J N, and HEADY, J A: Mortality in relation to the physical activity of work, *Brit. J Indust Med* 10 245, 1953
4. KARVONEN, M. J., KENTALA, E., and MUSTALA, O To be published.
5. KARVONEN, M. J., RAUTANEN, Y., and RIKKONEN, P To be published
6. KEYS, A., KARVONEN, M. J., and FIDANZA, F.: Serum-cholesterol studies in Finland, *Lancet* 2 175, 1958
7. KJELLBERG, S R, LONROTH, H, RUDHE, U., and SJOSTRAND, T: The relationship between the heart volume and the blood volume and its physiological and pathological variability, *Acta med scandinav.* 140.446, 1951
8. REINDELL, H *Diagnostik der Kreislauf-fruhschäden Physiologische Schwankungsbreiten, Regulationsstörungen und beginnende Schäden des Kreislaufes.* Stuttgart, Enke, 1949
9. RUOSTEENOJA, R., and LIND, J To be published.
10. PERE, S.: Clinical observations on leading Finnish athletes, *Duodecim* 61 388, 1948 (In Finnish.)
11. KARVONEN, M. J., RAUTAHARJU, P., RIKKONEN, P., and RUOSTEENOJA, R Heart size of champion skiers, *Ann med. int. Fenniae* 46 169, 1957.
12. HAKKILA, J: Studies on the myocardial capillary concentration in cardiac hypertrophy due to training: an experimental study with guinea pigs, *Ann med exper biol Fenniae* 33 (suppl 10) 1-82, 1955
13. RAUTAHARJU, P To be published
14. SIMONSON, E: Spatial vector analyzer for conventional electrocardiogram, *Circulation* 7 403, 1953
15. SIMONSON, E., and KEYS, A. The effect of age and body weight on the electrocardiogram of healthy men, *Circulation* 6 719, 1952.
16. CHRISTENSEN, E. H.: "Physiological Valuation of Work in the Nylorppa Iron Works" in FLOYD, W. F., and WELFORD, A. T. (eds). *Ergonomics Society Symposium on Fatigue* London, H K Lewis, 1953, pp. 93-108
17. ASMUSSEN, E., and NIELSEN, M Cardiac output during muscular work and its regulation, *Physiol Rev.* 35:778, 1955.
18. CHRISTENSEN, E. H.: Beiträge zur Physiologie schwerer körperlicher Arbeit Minuten-volumen und Schlagvolumen des Herzes während schwerer körperlicher Arbeit, *Arbeitsphysiol* 4 470, 1931.
19. JØRGENSEN, G: *Experimental Investigations of the Venous Pressure with Special Reference to the Regulation of the Circulation.* (Thesis, M.D., University of Copenhagen, 1953) Translated from Danish by E. R. Tottley Copenhagen, Danish Sc Press, 1954
20. NEUKIRCH, F: *Experimentelle Kreislaufversorgelser med særligt Hensigt paa Overgangen fra Hvile til Arbejde og fra Arbejde til Hvile.* Copenhagen, NYT Nordisk Forlag Arnold Busch, 1938.
21. ASMUSSEN, E., and NIELSEN, M: Cardiac output in rest and work determined simultaneously by acetylene and dye injection methods, *Acta physiol. scandinav* 27.217, 1952.
22. MELLEROWICZ, H: Vergleichende Untersuchungen über des Ökonomieprinzip in Arbeit und Leistung des trainierten Kreislaufs und seine Bedeutung für die präventive und rehabilitative Medizin, *Arch f Kreislauf-forsch.* 24:70, 1956
23. GREENFIELD, I.: Sodium succinate as test of circulatory efficiency, *Ann Int Med* 23 524, 1950

CHAPTER 24 WORK AND ENVIRONMENTAL STRESS IN THE CARDIAC FARMER*†

W. H. M. Morris

It is only very recently that any study has been made in the United States on the human stresses of farming. Our agricultural research in the past has been concerned mainly with crop production, animal production, marketing of agricultural produce, and similar items. The family farm is operated for the satisfaction of the farm family. Since about 98 per cent of all farms are family farms, the human factors connected with this satisfaction assume importance similar

to the physical factors connected with farm operation.

What, then, are the human stresses of farming and how do they differ from those in the industrial population, as shown, for example, by such studies as those of Hellerstein¹ and the United States Public Health Service at the Lockheed Aircraft Corporation at Burbank?²

Lack of Specialization

The first characteristic is the lack of specialization of labor in farming. In the first place the farmer is usually the capitalist, the manager, and the worker. He has to do a variety of work to maintain all his farm enterprises. Even with the trend toward specialization in fewer enterprises, the range of work is very great. Presumably, this precludes the nearly automatic operations that may occupy a factory worker for months, such as watching a machine make certain cuts or drill certain holes in thousands of similar parts.

*The studies described herein are part of the Purdue Farm Cardiac Project, a co-operative study, which is in part supported by the Indiana Heart Foundation and the American Heart Association. Journal Paper No. 1170 of the Purdue Agricultural Experiment Station.

†We have not yet reached the stage of being able to point out the specific differences in farm operation appropriate for farmers with differing degrees of cardiovascular impairment. These relationships are being studied in a survey, the field work of which has been completed, and in further studies to be made. After this we expect to have some of the answers but much will remain for future research.

Seasonal Variation in Load

Next, the work is highly seasonal in its type, in the amount to be done, and in the environmental stresses that accompany it. This certainly imposes a greater stress on the farmer than that due to lack of specialization. For example, in the spring in Central Indiana the month of April is often very wet, so wet that plowing is not possible on many farms. The farmer, highly motivated by the fact that he is working for his own family and for his family's gain, must do the plowing and disking at top speed in order to get as much corn as possible planted in the first three weeks of May.

The farmer's position in his community seems to depend somewhat upon his neighbor's assessment of his farming. This, together with his own personal satisfaction, is modified by whether he completes his farming operations before others, keeps the weeds controlled in his fence, and similar items. In the race to get the field work completed, quality must not be allowed to suffer; the rows must be straight and without many gaps. The Central Indiana farmer often has comparatively little work to do in March and sometimes in April, then suddenly he starts to work very long hours in the field, often in hot weather. He may be on the tractor from dawn to dusk and then return to the farmstead to do the chores. He may not leave enough time for sleep and in this way he adds to his stresses. In the past, when the farmer used horses, the horses controlled the number of hours he could work and obliged him to take rest pauses.

The time required for field work on Central Indiana farms then decreases to a minimum in August. Soybean harvest in September and corn harvest in October make a second peak in the annual field work curve. In December and January the field work becomes negligible. If the farmer has no chores, this is the time when he is free to take a vacation.

Heat Stress

The work on the farm can be divided broadly into field work and chore work. The

field work is not very arduous, judged by the metabolic cost, which averages about three Calories per minute (two and one-half times resting metabolic rate). However, field work must often be done in the heat of summer. We are studying the heat load on the farmers and find that on many days it is considerable even for a fit young man and severe for an older or impaired man. It is a common observation that hot humid days are uncomfortable and to be out in the fields working in such weather imposes a high environmental stress on the farmer.³ There are three main means by which the farmer can reduce the stress from the heat: a sunshade, a fan, and rest breaks.

He can use a sunshade to reduce the incident radiation from the sun. The black-globe temperature under a shade during the middle of the day averages seven to ten degrees lower than the unshaded globe temperature. This difference enables the farmer to drive his tractor with less heat stress than that of a man sitting still in a chair in the sun. This is the most simple and most effective way of reducing heat stress. Unfortunately, although sunshades are extensively used in the West, farmers in the Eastern and Central States seem to feel that their use involves a social stigma. This introduces a community education problem whose goal is to get older and impaired farmers to use sunshades.

The second means of reducing heat stress is by means of a fan, thereby increasing the wind speed and, consequently, the capacity to evaporate sweat. We have not tried this alone but by using a small fan with a sunshade during the middle of the day, the black-globe temperature can be reduced an average 30 to 50 per cent more than by means of the conventional sunshade alone.

It is also possible, as in industrial work, to schedule rest pauses to reduce the heat stress to a tolerable over-all level. However, on the farm there are no means of ensuring that the rest is taken. Since productive time is lost, this does not seem to be the most practical method. Needless to say, the rest should be taken in the shade. Future developments

may lead to air conditioning of the tractor cab or the tractor driver's clothing, which would reduce the stress to a tolerable level

Cold Stress

For the farmer who is fit the problem of cold stresses in the Central United States is apparently not serious. His activity produces heat and he can wear warm clothing. However, walking into a cold wind often brings on an attack of angina in a man with coronary heart disease. The only way that we know to avoid this trouble is to eliminate walking into the cold wind, either by changes in the layout of the buildings or by changing the farm operation to make it unnecessary to go out in this kind of weather.

Chore Work

The chore work is concerned largely with handling of materials, or moving feed, water, or animal products from one place to another. Many of the tasks are strenuous, for example, shoveling grain, loading manure, or carrying heavy loads. However, most of these tasks can be performed at a speed to suit the capacity of the worker. This enables the farmer to take a brief rest, if necessary, after throwing each scoop of corn or each fork load of manure.

Mechanization of handling of materials on the farm has reached a high stage of efficiency. In the poultry house, completely automatic operation has been accomplished. The feed can be ground and conveyed automatically to the poultry house. Automatic feeders and waterers can complete the elimination of moving the feed or carrying the water by hand. Automatic egg gathering devices are also available. The manure or droppings can be removed from the house by an automatic cleaner. For other types of livestock mechanical conveying of feed, self-unloading wagons, self-feeding the stock from the storage as, for example, with silage and hay, can reduce the hand work to a minimum. In milk production the cows can be fed with almost no hand labor. The stooping during milking can be eliminated by using a milking parlor with elevated stalls.

A pipeline milker can be used to eliminate carrying the milk and a bulk milk tank eliminates handling the milk in cans. Handling of poultry and hogs in confinement as opposed to permitting them to range in the fields also reduces the problems of handling materials.

Complete mechanization of the chore work is comparatively expensive. The capital requirements of our hog-corn farms and our dairy farms are already very high, about \$80,000 per farm among the Purdue University group of farmers keeping accounts. The extra capital required is often not available. However, much can be done on the farm by application of the principles of work simplification and a relatively small amount of additional capital.

Some strenuous activities still remain unmechanized, for example, castrating of young stock. The occasional temptation to run after livestock to head off the animals, to prevent breaking away from the herd, may be very dangerous. It is often done in hot weather and is almost always accompanied by emotional frustration. The combination of work, heat, and emotional stresses produces a severe strain. For a farmer who is a known cardiac such work will usually be out of the question.

Other Activities

There remains a group of activities that seems to cause greater distress to cardiac farmers than would be expected from the energy expended in their performance. These activities include driving fence posts, using an ax, and lifting heavy objects. They involve increased intrathoracic pressure and so increase the work of the heart. They must inevitably be performed in ballistic time, that is, the speed at which each cycle is performed cannot be controlled by the worker but is controlled by the tool or the weight lifted. For example, it does not reduce the stress of using an ax if the fall of the blade is controlled to a slow speed, or the stress of lifting if a weight is lifted by hand very slowly. The result of such control is to increase the forces required by introducing

static work to control the speed of performance.

CONCLUSIONS

The cardiac farmer faces problems somewhat different from the industrial cardiac worker, since the farmer usually supplies the capital, the management, and a substantial part of the labor for operating his business.

New techniques make it possible to substitute machinery for labor to a considerable extent in the operation of a farm. However, the capital requirement, which is already high, may become impossibly high if complete mechanization is achieved. Nevertheless, much can be accomplished to reduce the metabolic cost of farm work to match the capacity of moderately impaired farmers (New York Heart Association Functional Class I and II) by the use of work simplification principles and a relatively small amount of additional capital. However, there are certain farm jobs, such as running after livestock, which should not be undertaken by farmers with heart disease.

Apart from the stresses of work, the farmer is subjected to environmental and emotional stresses. Environmental stresses caused by heat can be reduced by sunshades, fans, and scheduled rest pauses. The emotional stresses, including those concerned with maintenance of social status, are among the most difficult problems facing farmers with heart disease. These stresses often drive the farmer to work beyond his capacity and for periods of time such that there is not sufficient time for sleep at night.

BIBLIOGRAPHY

1. HELLERSTEIN, H. K., and FORD, A. B.: "Energy Expenditure by Cardiac and Non-Cardiac Factory Workers," in *Work and the Heart*, New York, Paul B. Hoeber, Inc., 1959, Chapter 14.
2. USPHS. HEART DISEASE CONTROL PROGRAM: Unpublished material, 1957.
3. BELDING, H. S., and HATCH, T. F.: Index for Evaluating Heat Stress in Terms of Resulting Physiological Strains, *ASHVE, Journal Section, Heating, Piping, and Air Conditioning* 27:129, 1955.

CHAPTER 25 THE METABOLIC AND CARDIAC DEMANDS OF HOSPITAL AND REHABILITATION ACTIVITIES

*William G. Kubicek, Frederic J. Kottke, and
Jean N. Danz*

The rehabilitation of patients with heart disease presents multiple problems. Nearly all rehabilitation procedures require some degree of physical exercise. In some cases, physical exertion may be a hazard to the cardiac patient. The physician directing a rehabilitation program is then confronted with a set of conditions with rather poorly defined limits. Unnecessary caution prolongs the time for return to a normal life for the patient. On the other hand, even brief overexertion of the cardiac muscle may be dangerous in some cases. The physician must consider the complexity of the function of the cardiac muscle. Of the many distinguishing features of cardiac muscle, the resultant characteristic of automaticity can be used as a focal point of an experimental design. Superimposed upon the automaticity of cardiac muscle are the physical, chemical, and neuroregulating mechanisms. Of particular interest here is the neuromuscular system that provides a relatively quiescent

period followed by a sudden mobilization of millions of individual muscle fibers to provide a co-ordinated, efficient expenditure of energy by the cardiac muscle. The orderly spread of excitation over the heart muscle during systole is an important feature of the heart. It is an accepted fact that a damaged area in the heart muscle not only reduces mechanical efficiency but may also disturb the spread of excitation over the heart. It is conceivable that only a brief period of overload on a damaged myocardium could result in fibrillation. The rate of venous return and the filling pressure of the right heart are important considerations in this regard. On the arterial side, cardiac output and blood pressure determine the work load of the left ventricle. During equilibrium conditions, the over-all metabolic rate will determine the total heart work load to a large extent. Following abrupt changes in posture or rate of physical activity, transient disturbances may occur in the heart before regulatory mecha-

nisms can take over. For example, pooling of venous blood in the extremities and viscera is known to occur during periods of inactivity and especially while standing in the upright position. Abrupt changes in body position or physical activity can result in a transient but large increase in the rate of venous blood flow to the right heart. The resultant rise in right ventricular filling pressure, distention of the heart followed by the increase in left ventricular work load, should receive careful consideration by the physiatrist during the rehabilitation of cardiac patients.

PURPOSE OF INVESTIGATION

These experiments were designed to study the effects upon circulation, respiration, and metabolism of various hospital activities in the rehabilitation department. Two sets of conditions were used: (1) Steady state conditions after a prescribed physical activity had been in progress long enough for equilibrium to occur. (2) Transient conditions occurring during or immediately following changes in body position or degree of physical activity.

METHODS AND PROCEDURE

Two series of experiments were carried out: (1) Standard techniques for the measurement of oxygen consumption rate and cardiac output were used to study a variety of hospital activities. These methods provided data only after a steady state had been established for metabolic demand and cardiac output. (2) In a second series of experiments, electronic apparatus was used in order to observe some of the transient effects of certain hospital activities. An electronic computer was used to obtain a continuous record of oxygen consumption rate. Oxygen and carbon dioxide concentrations in the expired air, pulse rate, and arterial blood pressure were also recorded.

In the first series of experiments, studies were made on subjects in the fasting state. The subjects were young women students in occupational therapy. They came to the

laboratory after fasting since the previous evening, if the tests were carried out in the morning, or after fasting since breakfast, if the tests were carried out after noon. The subject rested for thirty minutes in a supine position on a standard hospital bed in a laboratory where there was a minimum of distracting noises. At the end of the rest period, oral temperature, blood pressure, and pulse rate were recorded. Oxygen consumption was measured, using either a McKesson Metabolar spirometer from which oxygen was inhaled or a Tissot type spirometer for collecting expired air for analysis. The subject breathed through a standard mouthpiece connected by a 6-foot tube of 1-inch internal diameter to the spirometer. The mouthpiece was supported by a yoke around the neck so that the subject could turn her head freely and carry on necessary activities while oxygen consumption was being recorded. If the expired air was collected in the large spirometer, it was analyzed for oxygen and carbon dioxide by the method of Scholander.¹ The oxygen consumption was calculated from the difference in concentration of oxygen and carbon dioxide between room air and expired air, and respiratory minute volume.

Cardiac output was calculated, using Grollman's acetylene method for estimating arteriovenous oxygen difference.² In calculating cardiac output, the solubility coefficient for acetylene of 0.00921 as determined by Chapman was employed.³ The acetylene rebreathing bag was prevented from collapsing by a 14-inch plastic tube of 1/2-inch bore and contained 42 holes that ran down the center of the bag from the mouthpiece.

For most of the estimations of cardiac output, alveolar air samples were collected from the rebreathing bag at the end of expiration after four breaths requiring nine seconds' time and at the end of fifteen seconds after three more breaths. Alveolar air samples were collected as indicated above for analysis of carbon dioxide, oxygen, and acetylene from which cardiac output could be calculated.

diac output at rest, the subject then assumed the position of the test and maintained it without doing work while metabolism and cardiac output were estimated again. The third measurement of oxygen consumption and cardiac output was made after the subject was working at a steady state on the activity under study. A period of at least 30 minutes was allowed between intervals of acetylene rebreathing to allow excretion of the acetylene which had been absorbed into the blood.

The following activities were studied:

Sitting Unsupported on Edge of Bed The subject was seated on the edge of a bed with arms in lap and feet supported on a chair

Getting out of and into Bed These data do not represent the peak load of oxygen consumption or cardiac output during this activity but rather the average throughout a 6-minute period of activity. A standard hospital bed 34 inches high was used for the study. The subject stepped to an 8-inch footstool and from there to the floor. Following a flashing timer, the subject carried out the following sequence of activities at 9-second intervals: (1) The subject rose from a supine position to sit on the edge of the bed. (2) The subject rose to stand on the footstool by the side of the bed. (3) The subject stepped from the footstool to the floor. (4) The subject stepped from the floor to the footstool. (5) The subject sat on the edge of the bed. (6) The subject returned to the supine position in bed. The above activity was carried out continuously through a 6-minute period used for recording oxygen consumption, followed immediately by a cardiac output determination.

Leather Tooling The subject tooled a standard design on leather while sitting in a bed with a Gatch frame, reclining at an angle of 45°. The bed table had the working surface inclined at an angle convenient for the subject. The subject rested her forearms on the table as she worked. A 3- by 3-inch piece of tooling leather that had previously been moistened and conditioned for tooling was marked with a conventional design. The

subject used a liner and spooner modeling tool to follow out this design.

Chip Carving The subject was seated in a straight backed chair at a table and used an Xacto knife with a ½-inch oblique blade for chip carving. She chipped ½-inch triangles according to a predrawn design from a 3- by 4-inch basswood block. A sound timer rang at 5-second intervals to control the rate of activity of the subject.

Platen Press Printing. The subject stood at a platen floor press that was operated with a foot pedal and printed 4- by 6-inch cards at the rate of 1 card per 5 seconds. In order to carry out the activity, the subject depressed the foot pedal that activated the press 4 times in each 5 seconds. As the platen approached the chase the first time the subject placed a card on the platen with the right hand and pushed the throw lever forward with the left hand. As the platen approached the chase the second time, the subject removed the printed card with the right hand and pulled the throwback lever with the left hand. This activity required the subject to balance on the left foot as she pedaled with the right foot and to use both hands in a co-ordinated sequence of activity of inserting the card, pushing the throw lever forward, removing the card, and pulling the throw lever back.

In the second series of experiments, the main emphasis was placed on developing methods to observe some of the transient changes in the circulatory and metabolic systems following abrupt changes in physical activity. The subject inspired room air. Through a valved mouthpiece, expired air was directed into a 1-inch hose connected to a large spirometer (American Meter Company). Respiratory minute volume was obtained from the spirometer movement, which rotated a small direct current generator. By proper calibration, the voltage output from the generator was related to the respiratory minute volume. A small diaphragm pump attached to a side arm on the hose between the subject and the spirometer diverted a continuous sample of the expired air into a Beckman Model F-3 Oxygen Analyzer* and

then into a Liston-Becker Infra-red CO₂ Analyzer,⁵ and then back into the spirometer. The output voltage from the CO₂ analyzer was connected to an Esterline Angus Recorder and also to a potentiometer network in series with the output from the O₂ analyzer. The resultant voltage from the O₂ analyzer and CO₂ analyzer was then recorded on a Brown Electronic variable span recorder, which also functioned as a servo-mechanism to operate a retransmitting slide wire, this then performed the multiplication of respiratory minute volume by the change in oxygen concentration between room air and expired air, corrected for CO₂ in the expired air. Arterial blood pressure was obtained by means of a small nylon catheter inserted through a thin-wall No 20 hypodermic needle into the brachial artery and attached to a Model P23D Statham strain gage, which was connected to a Sanborn

Poly-viso recorder. Pulse rate was obtained by a Waters Corporation Cardiometer, also attached to the Sanborn recorder. Oxygen consumption rate and respiratory minute volume were likewise recorded on the Sanborn Recorder. In these experiments running consisted of jogging in place at approximately two steps per second

RESULTS

Data obtained from the first series of experiments are presented in Tables 25-1, 25-2, and 25-3. Typical recordings of the data obtained with the electronic apparatus used in the second series of experiments are shown in Figs 25-1, 25-2, and 25-3.

Reclining in bed, sitting on the edge of the bed, sitting on a chair, and leather tooling in bed produced only a small increase in metabolic demand. Chip carving and getting

TABLE 25-1 THE INFLUENCE OF POSITION AND ACTIVITY ON THE METABOLIC DEMAND AS DETERMINED BY THE CONSUMPTION OF OXYGEN

Activity	Number of subjects	Number of tests	Mean metabolic demand cal./M ² /hr.	Standard deviation	Standard error of the mean	Metabolism percentage increase over supine
Supine	12	126	33.97	3.731	0.3325	
Reclining at 45° angle	6	60	35.06	3.373	0.4354	3.21
Sitting on chair	6	53	36.22	4.956	0.6820	6.62
Sitting on edge of bed, feet supported	3	19	37.70	3.849	0.8830	10.98
Leather tooling in bed, 45° angle	4	24	39.59	4.261	0.8676	16.54
Chip carving, sitting on chair	5	32	52.34	8.443	1.4924	54.08
Getting into and out of bed	3	17	56.86	4.224	1.0244	64.44
Printing press	5	21	82.05	10.349	2.258	141.54

TABLE 25-2 THE EFFECT OF POSITION AND ACTIVITY ON THE CARDIAC INDEX OF NORMAL YOUNG SUBJECTS

Activity	Number of subjects	Number of tests	Mean cardiac index liters/M ² /min.	Standard deviation	Standard error of the mean	Cardiac index percentage increase over supine
Supine	11	113	2.50	0.8746	0.0823	
Sitting on edge of bed	3	19	2.31	0.5432	0.1246	-7.60
Sitting on chair	6	50	2.33	0.4722	0.0668	-6.80
Reclining at 45° angle	5	46	2.69	0.4028	0.0594	7.60
Chip carving	3	32	2.89	0.6172	0.1091	15.60
Leather tooling in bed 45° angle	3	24	3.07	0.9516	0.1942	22.80
Getting into and out of bed	3	17	3.49	0.8750	0.2122	39.60
Printing press	5	21	4.25	0.9506	0.2074	70.00

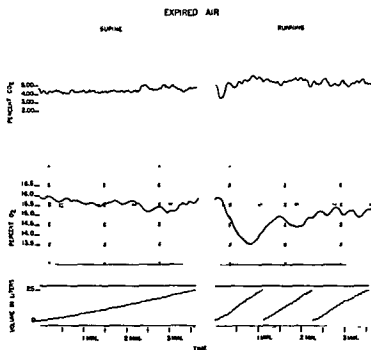


FIG 25-1 A continuous recording of volume of expired air, O_2 , and CO_2 concentrations in the expired air while a subject was supine and during running

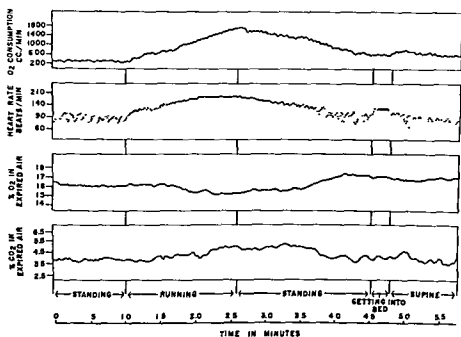


FIG 25-2 A continuous recording of O_2 consumption rate, heart rate, O_2 and CO_2 concentrations in the expired air while a subject was standing, running, standing, getting into bed and supine.

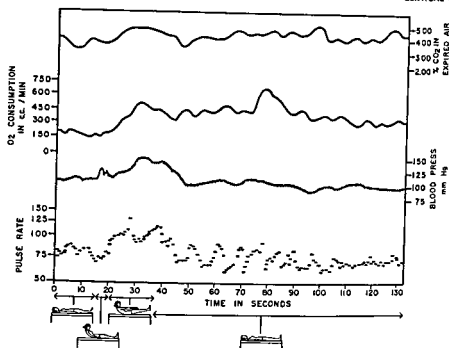


FIG 25-3 The effects of performing a *sit up* with arms extended horizontally and legs extended with the heels held six inches above the bed upon pulse rate, arterial blood pressure, oxygen consumption rate and CO_2 concentration in the expired air

into and out of bed imposed a moderate but distinct increase over basal in metabolic requirement, while operating the printing press more than doubled the energy required over basal (Table 25-1)

Table 25-2 shows the results of the studies described above on the effect of body position and physical activity on cardiac index. Of interest here was the apparent decrease in cardiac output when the subjects changed from the supine position to sitting on the edge of the bed or in a chair. The data indicated small increase in cardiac output when the subjects changed from the supine position to reclining in bed at 45° with the knees flexed. Moderate but more definite increases in cardiac output of 15 to 20 per cent over supine were observed during chip carving and leather tooling in bed. Getting into and out of bed resulted in an increase in cardiac output of approximately 40 per cent and operating the printing press imposed the largest load with a 70 per cent increase in cardiac output.

Table 25-3 provides a convenient means of comparison of the data from Tables 25-1

and 25-2. The activities studied are arranged from top down in order of progressively greater metabolic demand. For these same activities, the cardiac output studies yielded

TABLE 25-3 A COMPARISON OF METABOLIC RATE AND CARDIAC INDEX

Activity	Metabolism percentage increase over supine	Cardiac index percentage increase over supine
Reclining at 45° angle	3 21	7 60
Sitting on chair	6 62	-6 80
Sitting on edge of bed feet supported	10 98	-7 60
Leather tooling in bed 45° angle	16 54	22 80
Chip carving sitting on chair	54 08	15 60
Getting into and out of bed	64 44	39 60
Printing press	141 54	70 00

rather inconsistent data, since the changes in cardiac output were not proportional to the increases in metabolism until the metabolic rate had been increased over 60 per cent by

getting into and out of bed or by operating a printing press

From the second series of experiments, continuous records of volume of expired air, O_2 and CO_2 concentrations in the expired air, with the subject supine in bed and then running, are presented in Figure 25-1. Respiratory minute volume was calculated from the slope of the line indicating volume. The main points of interest here are the transient changes in O_2 and CO_2 concentrations in the expired air during the first minute of running. In spite of a sharp increase in respiration, O_2 concentration fell from 16 per cent to 13.5 per cent in the expired air during the first minute of running. After about $2\frac{1}{2}$ minutes of continued running, O_2 and CO_2 concentrations in the expired air stabilized near the control levels.

The recordings in Figure 25-2 illustrate data that do not show any unusual transient changes at the beginning of exercise but do indicate the length of time necessary for a steady state to be reached, following an abrupt increase in physical activity. Heart rate and O_2 and CO_2 concentrations in the expired air stabilized in about one minute while O_2 consumption rate was still increasing when the subject stopped running after $1\frac{1}{2}$ minutes of running. The procedure of getting into bed was accompanied by an increase in pulse rate followed by an increase in O_2 consumption rate. Pulse rate did not vary during the respiratory cycle during exercise, while obvious changes with respiration occurred during the rest periods.

The data in Figure 25-3 illustrate the rate and extent of changes in pulse rate, arterial blood pressure, oxygen consumption, and CO_2 concentration in the expired air while a subject did a partial sit up, followed by a period of sitting up with the arms extended horizontally and the legs extended with the heels held about six inches above the bed. When the subject changed from the supine to the sit-up position, only a small transient increase occurred in the blood pressure. Holding the arms extended and heels off the bed for eighteen seconds produced increases in all the variables recorded. On the

average, pulse rate increased from approximately 80 to 115 beats per minute, blood pressure rose from 100 to 150 mm. Hg, oxygen consumption increased from 160 to 460 cc/M²/min and CO_2 concentration in the expired air increased from 4 per cent to 5 per cent.

DISCUSSION

The rehabilitation of a handicapped individual usually requires some type of physical activity. The rehabilitation of cardiac patients presents a challenging medical problem since overexertion may be dangerous to the patient and undue caution can hamper the rehabilitation program.

Relatively little study has been devoted to determining the effect on the heart of various types of normal hospital activities. Most studies have been made on normal or abnormal hearts during rest or some standardized condition of exercise. Because of technical requirements during the measurement of cardiac output, it has been difficult to test the various activities carried on by patients in the hospital or persons leading sedentary lives. The physiologists interested in the work of the heart have wanted to know the range of cardiac capacity. The cardiologists have usually been more concerned with the problems of valvular inefficiency or demonstration of abnormalities than in attempting to control cardiac work by regulating activity in a quantitative manner.

Recently it has been advocated that cardiac patients be allowed to sit in a chair early in the course of recovery from coronary thrombosis^{6,7,8,9}. Although this has been viewed with alarm by some physicians, the statistics for this method compare favorably with prolonged bed rest. The data on cardiac output in this and previous studies indicate that there is less cardiac demand when the patient is sitting with his feet dependent than when he is supine^{2,6,10,11}. Clinical observation also supports this finding. The patient with cardiac embarrassment wants to sit with his feet dangling. To

place such a patient in a semireclining position with the back rest and knee rest up may decrease his muscular activity very slightly but increase his necessary cardiac work by as high as 15 per cent. Minimal cardiac and metabolic demands occur when the patient is seated in an armchair with back and head supported and the feet comfortably supported on the floor. The data in this study of the cardiac demand of moving without aid from bed to chair and back indicates a higher cardiac requirement than would be the case if the patient were assisted to sit and to stand. Although these latter measurements have not been made, it is likely that movement from bed to chair, if done carefully and with assistance, places little demand on the heart.

It is a maxim of medicine that an injured organ should be put at rest so that it may recover more quickly. For many years it has been assumed a priori that the recumbent position is the position of least activity for most or all organ systems. This position is referred to as basal, although that term in actuality applies only to oxygen consumption by the voluntary muscles. The orthopnea of the cardiac patient is usually explained on the basis of changes in the mechanics of respiration rather than on the work requirements of the heart itself. It now appears that the orthopneic position is the position of minimal cardiac work as well as the position of greater ease of respiration.

Although it would be desirable to maintain the severely damaged heart at a condition of minimal or basal work for a number of days to allow recovery, this is not possible in all cases. The activities of digestion and elimination must continue. Emotional stresses due to fears about illness or death, concern over job, family, or future must play a role in determining the level of cardiac output.^{8,12} The patient will move in bed to drink or eat, from discomfort, boredom, or anxiety. If he is maintained on enforced complete rest, these factors probably become more prominent and as a result the cardiac work increases.

For bed-bound patients a change is a rest

in that it allows relaxation to occur. To move the patient carefully from bed to a comfortably supporting armchair places the patient in a physical position of minimal heart work and also helps to relieve emotional tension. The chair position assumed several times a day, therefore, keeps the cardiac output closer to the minimal level than does absolute bed rest.¹³ To assist in relieving emotional tension and anxiety, diversional activities such as reading or occupational therapy crafts requiring the use only of the hands are valuable. The activity allows the patient to forget his problems and results in a decrease of tension. The lightest crafts, which require use only of the hands, increase the cardiac requirement about 15 to 20 per cent. It is probable that anxiety exerts a greater driving force than this on the heart. Both the drive of muscular effort and the drive of emotional stress must be avoided if the heart is to be protected against work. During convalescence the gradual addition of activities requiring progressively greater cardiac work appears to be the logical way to restore the myocardium to normal levels of function. During this period of recovery, controllable activities of known metabolic and cardiac demand are valuable to help to condition the heart. Since activities in occupational therapy can be quantitated and controlled, they appear to be ideal for the purpose of rehabilitation of cardiac patients.

SUMMARY

The cardiac demands of bed activities and light activities in occupational therapy were studied on normal subjects. The cardiac output while sitting in a chair or on the side of the bed was slightly lower than that when lying supine, and the cardiac output when reclining with the back rest and knee rest up was slightly increased. The cardiac requirement to get out of or into bed showed high and low transients when carried out unassisted, but this activity probably does not demand a great deal of myocardial work if done slowly with assistance. Occupational therapy using primarily hand motions in-

creased cardiac output very little. Standing to use a printing press increased cardiac output 60-70 per cent. These activities, because they can be controlled, offer an excellent means of gradually reconditioning patients with cardiac disease.

The use of electronic apparatus for rapid, continuous, and simultaneous recording of several variables concerned with circulation and metabolism offers an improved approach to the study of transient changes that may occur in the circulatory system before the regulating mechanisms can gain control. The data indicate that an abrupt change in body position or in level of physical activity can produce a rather large increase of short duration in cardiac work. Changing position or rate of physical activity slowly and with assistance can reduce this type of stress on the heart.

BIBLIOGRAPHY

- 1 SCHOLANDER, P. F.: Analyzer for accurate estimation of respiratory gases in one-half cubic centimeter samples, *J Biol Chem* 167 235, 1947
- 2 GROLLMAN, ARTHUR: *The Cardiac Output of Man in Health and Disease* Springfield, Ill., Charles C Thomas, 1932
- 3 CHAPMAN, C. B., TAYLOR, H. L., BORDEN, C., EBERT, R. V., and KEYS, A.: Simultaneous determinations of the resting arteriovenous oxygen difference by the acetylene and direct Fick methods, *J Clin Invest* 29 651, 1950
- 4 PAULING, L., WOOD, R. E., and STURDIVANT, J. H.: An instrument for determining the partial pressure of oxygen in a gas, *Science* 103 338, 1946
- 5 WILLIAMS, W. Z.: Infra-red instrumentation and techniques, *Rev Scient Instruments* 19 135, 1948
- 6 COF, W. S.: Cardiac work and chair treatment of coronary thrombosis, *Ann Int Med* 40 42, 1954
- 7 LEVINE, S. A.: Some harmful effects of recumbency in the treatment of heart disease, *JAMA* 126 80, 1944
- 8 LEVINE, S. A., and LOWN, B.: "Armchair" treatment of acute coronary thrombosis, *JAMA* 148 1365, 1952
- 9 BECKWITH, J. R., KERNODLE, D. T., LEHEW, A. E., and WOOD, J. E., JR.: The management of myocardial infarction with particular reference to the chair treatment, *Ann Int Med* 41 1189, 1954
- 10 ASMUSSEN, E., and NIELSEN, M.: Cardiac output during muscular work and its regulation, *Physiol Rev* 35 778, 1955
- 11 GROLLMAN, A.: The effect of variation in posture on the output of the human heart, *Am J Physiol* 86 285, 1928
- 12 STEAD, E. A., JR., WARREN, J. V., MERRILL, A. J., and BRANNON, E. S.: Cardiac output in male subjects as measured by technique of right atrial catheterization: normal values with observations on effect of anxiety and tilting, *J Clin Invest* 24 326, 1945
- 13 KOTTKE, F. J., KUBICEK, W. G., DANZ, J. N., and OLSON, M. E.: Studies of cardiac output during the early phase of rehabilitation, *Postgrad Med* 23 533, 1958

REPORT OF PANEL ON CLINICAL PHYSIOLOGY

Howard B. Burchell

The purpose of the panel on clinical physiology in this Conference has been to identify and validate the physiological parameters that would define the efficiency of the circulation in subjects with established or suspected heart disease under various degrees and types of stress. Although the criteria for the diagnosis of heart disease well might have engaged our attention, this problem appeared secondary to inquiries into the functional status of the circulation in persons of an aging population who do or do not have symptoms of heart disease.

In the initial planning for this panel, some basic questions were developed to highlight the various discussion periods to be held during the Conference. These were (1) Does work have any effect upon the development of heart disease, specifically, coronary atherosclerosis; (2) What environmental factors in association with work produce stress upon the heart and upon the circulation; (3) What is the value of physical fitness tests in classifying individuals regarding the severity of their heart disease? Its early recognition?

work loading for an individual? While the answers to these three questions could not have been expected to be simple ones to which everyone could subscribe unequivocally, there appeared to be reasonable agreement within the panel regarding tentative conclusions as to the direction in which investigation may proceed with best advantage.

Dr A. Keys reviewed the relationship of environmental stresses to heart disease and pointed out the weaknesses in the studies of populations which are believed to support the concept that exercise protects the individual against the development of clinical coronary artery disease. If this protective effect is present, as certain animal experiments and clinical impressions suggest, more extensive studies are necessary to establish its existence.

In respect to occupational groups, Dr W. H. M. Morris presented figures indicating that there is a high incidence of coronary disease among Indiana farmers, but that clinical appearance of the disease definitely

lation The relationship of work and the cholesterol level in the blood was put to experimental tests by Dr H L Taylor. He reported that if the cost of the work was paid by food supplements having the same distribution of basic foodstuffs as the basic diet, no significant changes in levels of serum cholesterol occurred

Dr S. G. Wolf reported on the effect of varying life situations on serum cholesterol and reported that increments of serum cholesterol level regularly followed series of psychic storms These storms were recognized and evaluated well only by careful interviews wherein there were favorable patient-physician relationships It appeared that much more work may be done profitably in this investigation of the influence of various environmental factors upon serum cholesterol levels

The presentations of many of the panelists, and particularly those of Dr L A Brouha and Dr W H M Morris, repeatedly brought three facts into clear focus: (1) The actual work load upon the worker in industry and on the farm, as determined by oxygen demand, is usually not heavy and can be expected to be tolerated well by persons with limited damage to their hearts, (2) The cardiac stress that is reflected in maximal heart rates is not necessarily dependent upon the nature of the work nor is it correlated simply with the cost of work as measured by supplemental oxygen utilization, (3) The stress of a total work situation is often to be found not in the work per se but in such factors as elevated temperatures or high humidity Although less susceptible to techniques of measurement, social pressures and psychic adjustment to work situations are recognized as important facets of the problems presented in this field

The joint meetings of the panel on basic physiology with that on clinical physiology brought out presentations and discussions that led to some clarification of definitions and concepts of impaired function of the heart and circulation It appears that there are still many aspects of physical fitness that escape identification While there are various

tests that reflect the capacity of the patient to do work, they do not seem adequate in grading individuals whose cardiac disability is in its inception nor in the prediction of difficulties about to be manifest The value of simple, well-standardized tests need not be re-emphasized to this group It is notable that during the discussions it appeared that there was considerable interest in the revival and re-examination of several older techniques, including measurement of oxygen debt following standard exercise and the response of the circulation to sustained high intrathoracic pressure as in the Flack test and the Valsalva maneuver In the joint meeting it was emphasized that there is need for differentiating total physical fitness from its cardiac component The studies of Dr Bruce seemed to form one excellent basis for future investigations along these lines

If the panel on clinical physiology had been empowered to bring resolutions to be passed upon by this Conference, I might suggest the following, provided that my fellow panel members had the opportunity to make corrections and revisions (1) It is necessary to make further observations regarding the role of various occupations in the development of heart disease; (2) In any work situation being evaluated by the governing agencies, it is important to study the total environmental stresses involved and not to be limited to the determination of the work load that the task alone imposes upon the worker, (3) There is a need for further evaluation of indexes of physical fitness and measurements of physical endurance This Conference might do well to endorse an international effort to study this problem; (4) The problems of cardiac efficiency and that of the total circulation in disease and aging need new approaches Any scheme that brings together the scientists in the field of basic physiology and the clinicians should produce worthwhile results In our discussions we have learned to speak not only of work accomplished by the heart but of power This concept of work as opposed to power may be among the most pertinent accomplishments of this Conference

I am grateful for the contributions made by the various members of the panel on clinical physiology as well as those of my co-moderator, Dr Charles Crumpton, and the recorders Dr A Keys and Dr. G. Biörck have given some special attention to the problem of physical fitness. They have developed some suggestions, which I would like Dr Keys to present to the Conference as a supplemental report from our panel.

DR ANCEL KEYS: As Dr Burchell has indicated, there are some points pertaining to physical fitness which seem of such consequence to some of us and so pertinent to our discussion here at this Conference that we devoted some hours to these problems last night. Dr. Biorck was good enough to act as a recorder during that meeting and we have some notes which I will summarize briefly. The participants in this discussion included Drs Katz, Taylor, Hellerstein, Warren, Eichna, Karvonen, and Morgan Jones. We met as a small group simply as persons who had in one way or another devoted time to or done actual work on the problem of physical fitness. We agreed that in any discussion of work and the heart, the matter of estimating fitness and defining what you are attempting to estimate is of great concern. We felt, in our meeting, that there has been a great deal of confusion in this Conference and that a similar confusion exists in the literature because the phrases *fitness tests* and *fitness* are used in different connotations. Moreover, we feel there is a need to develop quantitative, objective methods that can be used as an aid in diagnosis, to provide an aid in prognosis as to the future course of the disease, to guide the adjustment of the patient to his job and to his life situation, and to measure the response of the patient to the regimen and program of management arranged by his physician. It was agreed that every effort should be made to encourage objective evaluation of tests and the exchange of information regarding them. We hope this Conference will go on record endorsing these general principles and calling the attention of organizations of national and international character to this

problem that they may put into operation research programs, exchanges of information, and forward-going planning along the lines already outlined.

DR. BELKNAP: Thank you very much, Dr Burchell and Dr. Keys. We would like to have some discussion from the floor on both of these reports.

DR. LUCIEN BROUHA: I would like to add a brief word to what Ancel Keys just said, re-emphasizing the need for specific fitness tests and also for a better definition of what we mean by *fitness*. What I have in mind is that one of the problems that we face daily in industry is to find out which is the best test to use to determine whether or not a worker is fit for the job that he is doing. This is not the particular aspect of the field of physical fitness that we investigated during the war when we were concerned with physical fitness for extreme effort or exertion. We should attempt to develop tests that are applicable to men of various ages and will help to determine whether or not these individuals will be able to fulfil the job for which they are being considered. I believe that a great effort should be made in this line in order to help solve the problem of preplacement examination and selection of men for the right kind of job.

DR. JAMES V. WARREN: It seems to me that we have at least three considerations here. One of the problems in the literature and in various laboratories, I am sure our own laboratory, is that these get confused at times. We have the problem of training, or physical fitness, or, as we see it, adaptation, in an athlete for maximum performance. We have the problem of evaluating the effects of disease on the normal function, whether a person is in the level of training or is untrained. Finally, we have the consideration of prognosis, which sometimes gets involved in this, and, as pointed out by Dr. Hellerstein yesterday, a test will often show perfectly normal findings but give no idea of a serious prognosis. I believe it would be of great benefit, in the future, to point out clearly what these tests are aiming at in these three areas and, probably, in others as well.

DR. RODNEY BEARD: The discussion in the Workmen's Compensation Panel touched upon this question of testing very briefly, when one member asserted that there is no test that is satisfactory except the test of return to work. Now, I do not believe that he implied or meant to imply by this that additional tests were not desirable. But I do believe that the members of our panel would take a very dim view of the prospect of developing satisfactory tests because of the very great complexity of psychological factors that enter into the performance of an individual under test circumstances.

In particular, of course, when one comes to the question of disability, motivation becomes a very strong factor, but it is not alone in this circumstance that motivation is involved. I would like to hear further comment on this point from Dr. Klein.

DR. IRVIN KLEIN: Our experience at the Bellevue Hospital Work Classification Unit and at the Beckman Hospital Unit indicates, at least to us, that no scientific test is of any great value in determining either the prognosis or the workability, because the test, and let us assume it is the treadmill, does not consider many of the human factors that operate in influencing the determination.

We have felt for some years that the return to work is probably the best test we have, because it automatically, and without our knowledge or control, takes into consideration many of the psychological and human elements concerned. I do not believe these factors can be measured in such a way that we can prepare a chart, and, if we cannot do that, there are certain things in disability evaluation and workmen's compensation that I feel will never be measured. It is the feeling in our clinic that these things can be measured only by putting the man back into as near a normal state of affairs as we can.

DR. WILFORD H. M. MORRIS: I would like to make two comments. First, that I believe the tests we have been discussing are tests of impairment and not tests of disability. Clearly, what Dr. Klein is requesting is a test for disability. I believe this is something

that is beyond the physiological approach or probably even beyond the clinical approach.

Secondly, I believe that an added use for the type of fitness test we have been discussing is in research. An example is the study of the improvement or the change in fitness resulting from cardiac surgery. In addition, as in our own case, a fitness test on cardiac patients may be valuable for comparison with cardinals studied in other laboratories.

DR. GUNNAR BJÖRCK: I believe it is necessary to state that there are at least three purposes of such tests. One, relating to basic physiology of normal and pathological situations; the second, referring to early detection of disease and the determination of prognosis; and the third, relating to the determination of fitness for certain jobs. I believe it is necessary that we divide our thinking along these lines. At the small meeting to which Dr. Keys has referred, it was felt that there was a great need to investigate the tests that have been used along these various lines, and that every test, particularly in clinical situations and with regard to job orientation, should be evaluated later on according to its purpose. Those tests which possess a prognostic aspect should be carefully re-examined after a series of years by means of follow-up studies to ascertain the predictive value of the tests. Research workers should be encouraged to make and report such studies.

DR. MARTTI J. KARVONEN: The physiological barometer of training needs measurement and this is one of the obvious uses for fitness tests, both in the healthy person and in the cardiac. Measurement of this barometer cannot be achieved in any other way, so fitness tests are needed.

DR. LUDWIG W. EICHNA: It is my belief that some confusion has entered this discussion. I would like to submit that there is a clear distinction between a test for cardiovascular fitness and a fitness test for an individual. There are tests that determine the fitness of the heart and the circulation. This does not mean that this is the sole parameter of an individual's fitness. It is one of the

parameters of his fitness that can be determined. The result of such a test should be given to the physician who then must evaluate whether the individual is fit in other categories, including his motivation, psychologically, and so forth. Unless this is done, there will be confusion.

DR. HOWARD B. BURCHELL: I welcome this discussion in the field of physiology. It is somewhat belittling, perhaps, to my report in that I felt I had spelled out some of these things. I believe I pointed out very clearly that we were measuring different things and we were approaching the problem from different angles.

I do remember that Lord Kelvin said that when you can measure something, you can begin talking about it. It is for this reason that we do have to have some measurements. In the clinic, at the present time, with our emphasis upon the clinical history, we see histories written, physical examinations performed, and opinions given, only to go in to see the patient in consultation and find that he has not been able to exercise at all. A simple test in the clinic, such as having the patient step up and down a few times, makes clear the degree of limitation of the patient. There are certain tests that have been pointed out as quite specific, and outside the cardiac field we can find some preliminary tests that are quite specific in regard to early illness. Now, if one wants a test to evaluate a person for work, I believe that the observations reported at this Conference have already indicated that such special tests are hardly necessary. It was shown by Dr. Hellerstein and his associates that the actual work per se was not usually a major factor but that there are other factors, many of them environmental, which are of great importance.

DR. LEWIS H. BRONSTEIN: Since the Bellevue and Beekman Hospital Work Classification Units have been mentioned, I would like to clarify some of Dr. Klein's remarks. In the seventeen years that Dr. Goldwater, Dr. Klein, and I have been working, trying to get the patients with heart disease back to work, we have not been able to

find any test that has helped very much. At present, we feel that our clinical evaluation based upon the history and physical examination and our observations of the patient at work are probably the best measures we have. However, we do not wish to intimate that further efforts should not be made to get some objective measurements that can be interpreted, as Dr. Eichna has said, in the light of the rest of the picture. We do have the fear that some persons are going to try to use the tests and forget to use their clinical judgment and evaluation. This is the point that we wish to emphasize.

DR. HERMAN K. HELLERSTEIN: We have studied 1,200 patients in Cleveland after the fashion prescribed by Dr. Goldwater. We have found that a total evaluation of the person in terms of medical, social, vocational, occupational, and physiologic parameters is extremely valuable. On the other hand, we have had considerable help in several ways from the objective evaluation of the cardiovascular function. We have done the Flack test and we have done various tests in association with Dr. Ford concerning the energy requirements of physiologic loads of known or measured value. In addition, we have tried to correlate these results with on-the-job evaluation and with studies during

tests is to be decried. It has been extremely valuable to know that some persons with different types of heart disease sometimes respond in a way that is better than clinical methods tend to indicate. I have great respect for the use of the eyes and ears, and, having been under the influence of Dr. Katz and many others in my early formative period, I believe in the value of the physical examination and a good clinical evaluation. However, we do not have eyes that can detect electrical changes and other physiological phenomena. I would make the plea that a nihilistic attitude regarding the use of tests in this field be avoided.

DR. ROBERT BRUCE: I feel I may be, in part, responsible for some of the confusion

that seems to have developed in the course of these deliberations about the relative importance and significance of physical fitness testing. Perhaps this is an inevitable error that comes about in trying to set down briefly what we have been doing. It may be important to emphasize, again, how we try to use this methodology and how we try to interpret data from it.

I think that we should say, first of all, that we are interested in evaluating cardiac impairment of patients with heart disease. This means that we do have a physical examination, history, and the usual clinical evaluation, which is as complete as possible prior to testing. It does not mean that we are trying to suggest that a test will take the place of the clinical appraisal of the patient under any circumstances. This is clearly not the case, but we do feel that it may give more depth to the usual physical examination if we have the opportunity of examining patients under some type of stress that can be relatively and conveniently standardized for the greatest number of patients that we

have the opportunity of studying. In this sense the procedure I have been discussing, and which has led to some of the confusion here, has been useful in our laboratory for some period of time.

The second point is that I believe there is a great need for developing a greater understanding of the physiological changes of cardiac patients in relation to stressful situations. Exercise is one of these though not necessarily the only one. We have been considerably intrigued by the opportunity to get more information along this line so that we will have a better understanding of cardiac physiology and its application to clinical problems.

Now, the error that may come is to try to read too much into a simplified laboratory procedure and to expect that from it we can extrapolate all the things we would like to know about functional capacity and fitness in the broad sense, all of them things that take in much more than just the heart alone, as well as the possibility of prognosis.

PART III PATHOLOGY

CHAPTER 26 INTRAMURAL HEMORRHAGES AND THE HISTOGENESIS OF ATHEROSCLEROSIS

John D. Hamilton and J. H. Mowbray

Some time ago, we began to study the coronary arteries in cases of myocardial infarction and coronary thrombosis with a view to determining how frequent or infrequent was the association of intramural hemorrhage with thrombosis, and what relationship could be established between effort and intramural hemorrhage with or without thrombosis. With these objectives in view, the coronary arteries were examined from 100 unselected consecutive adult autopsies performed in the Department of Pathology of the Toronto General Hospital in 1955. In this series there were 22 deaths attributable to coronary artery disease.

Technique

All 100 cases were studied by a slight modification of a technique first applied to coronary arteries by Durlacher.¹ In brief, the heart is removed intact, and the coronary arteries perfused by gravity via their

ostia with saline followed by 10 per cent formalin. After 48 hours fixation the vessels are dissected from the heart down to the smallest accessible surface branches (approximately 1 mm in diameter). They are then dehydrated, cleared in methyl benzoate, and examined in the clearing solution (Fig 26-1). Hemoglobin-containing lesions stand out as reddish-black masses (Fig 26-2). Blocks are taken of all hemorrhages, areas of narrowing, and occlusions. In those cases in which hemorrhages are numerous, only representative samples are taken.

Microscopic sections were cut at 5 μ . Serial sections of many hemorrhages and thrombi, but not all, were cut. At times, especially with extensive lesions, about 50 sections were examined every 0.5 mm. through the area. The method of sampling lesions and of taking microscopic sections should be kept in mind in assessing our results. All histologic sections were stained

TABLE 26-2 MICROSCOPIC GRADING OF ATHEROSCLEROSIS

Feature	1	2	3	4
Lipid deposit	Streaky, small \pm	Larger, but no amorphous material +	Amorphous pools ++	+++
Cholesterol clefts	0	0	+	+
Vascularization	0	Slight, marginal only	+	+
Hemorrhages	0	0	+	++
Hyalinization	Thin, surface only	Thicker, surface and margins	Forming crescents	Usually multiple crescents
Media	Normal	Normal or slight thinning	Atrophic	Atrophic and disrupted

Recognition of hemorrhages microscopically depended upon identifying red blood cells. This means that the hemorrhages were recent and fresh although it was possible in some cases to postulate that there had been more than one episode of hemorrhage, judging by the state of preservation of red cells

TABLE 26-3 DISTRIBUTION OF ATHEROSCLEROSIS ACCORDING TO GRADE

Grade	Cases
I	11
II	14
III	26
IV	37
Total	88

and leucocytes. In the majority of cases exhibiting hemorrhages, a period of hypotension or anoxia could be found in the recent history, but on the other hand, similar episodes could be found in the cases not showing hemorrhages. There was evidence, in some of our cases, that intramural hemorrhages were larger and more frequent distal to a recent thrombus. However, the only feature that could be definitely correlated with the intramural hemorrhages was the grade of atherosclerosis. With increasing size of amorphous pools of lipid and increasing vascularization, there was an increasing incidence of intramural hemorrhage. There was no correlation with anticoagulant therapy. Evidence of old hemorrhage, in the form of hemosiderin pigment, was infrequently encountered with our method of examination.

Source of the hemorrhages could not be determined, except in some of the cases of thrombosis when the hemorrhage was massive. As stated above, vascularization of plaques was seen in Grades II, III, and IV atherosclerosis. This derived from vasa vasorum in the majority, as transmural channels were frequently and easily seen. In 11 cases luminal origin was noted on routine sections. In 25 other cases, however, position of the vessel made luminal origin obligatory. In these latter cases such vessels lay separated from the lumen by little more than a strand of connective tissue and two layers of endothelium. Such a channel must carry blood under pressure equal to that in the lumen in order to maintain its patency. In about half the cases showing vascularization of plaques, then, vessels taking origin from the lumen contribute to the vascularity (Figs. 26-3, 26-4, and 26-5).

TABLE 26-4 INCIDENCE OF INTRAMURAL HEMORRHAGES

Atherosclerosis, grade	Number
I	0
II	0
III	16
IV	35
Total	51

THROMBOSIS. There were 14 cases in which recent coronary thrombosis was found. These thrombi were studied by serial and/or interrupted sections throughout their length



FIG. 26-3 Large intimal channel arising from the lumen

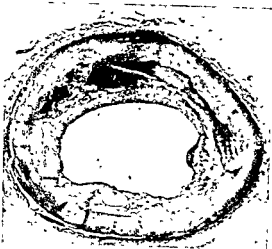


FIG. 26-4 Large intimal channel in loose areolar zone. A vessel so located must communicate with the lumen (confirmed by serial section). Note the external incomplete ring near media. Intimal hemorrhage is present. $\times 12$

In all cases the thrombi overlay atherosclerotic plaques. Table 26-5 indicates that intramural hemorrhage was found in the underlying atheroma in all but two instances. Frequently the superficial layers of the plaque immediately beneath the thrombus were necrotic. In this table, *inconsistency* means that the oldest part of the thrombus did not overlie the area of maxi-

mal hemorrhage, and in seven cases the oldest area of thrombus was not associated with the area of maximal intimal hemorrhage. In three cases there was an association between thrombus and hemorrhage. Rupture of the atheromatous plaque was

TABLE 26-5 RECENT CORONARY THROMBOSIS

Number of cases	Intramural hemorrhage	Necrosis of plaque	Rupture Inconsistency of plaque	
14	12	8	7	4

found four times, and was always associated with massive hemorrhage in the atheroma. In serial sections, rupture usually occurred at more than one point. Our interpretation of the massive accompanying hemorrhage is that it derived from the lumen and was in the nature of dissection of the plaque (Figs 26-6, 26-7, and 26-8).

Discussion

Although intimal blood vessels have been recognized for many years,³ the work of



FIG. 26-5 Endothelium-lined channels (central, large) lying superficially in the intima. Subsequent sections showed a plexus communicating with the lumen. $\times 240$.

Paterson,^{4a,b} Winternitz,⁵ Wartman,⁶ and Geiringer⁷ has stressed their importance in the development, progression, and sequelae of atheroma. When one comes to consider the origin and development of these vessels, there is no unanimity of opinion. Winternitz implies, but does not state, that they may be found normally in man, but he has described them in the normal aorta of the cow. Paterson has demonstrated vascular channels in normal intima, but they are exceedingly rare. I think one may question the presence of blood vessels in the intima as a normal and usual occurrence, but certainly there is uniform agreement that the intima thickened by atheroma is vascularized, the vessels taking origin from the vasa vasorum and from the lumen. Frequently there is anastomosis between the two. The most logical explanation for those vessels arising from the lumen is given by Duguid,⁸ who has developed the thesis originally advanced by Rokitsansky,⁹ that an atheroma is produced by deposition of fibrin of the arterial blood. In other words, mural thrombi form, become organized by endothelium, and incorporated into the intima. It is the remnants of this organization that persist as the vasculature derived from the lumen. Geiringer goes as far as to say

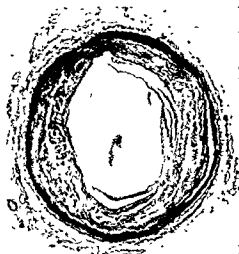


FIG 26-7 Intimal hemorrhage. Dark areas represent hemorrhages of different ages in same segment. The one at the left shows some organization (see Fig 26-8) $\times 12$.



FIG. 26-6 Intimal hemorrhage. Entire amorphous mass contains hemorrhage mixed with atheromatous material. On gross examination and on superficial microscopic inspection the hemorrhage appears to have caused occlusion, but in reality it has not altered the lumen.



FIG 26-8 Intimal hemorrhage. High power to show large irregular vascular channels at the margins of area of hemorrhage $\times 240$.



FIG 26-3 Large intimal channel arising from the lumen

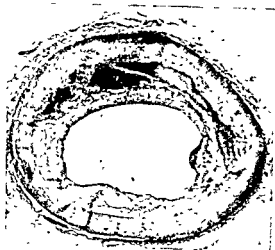


FIG 26-4 Large intimal channel in loose areolar zone. A vessel so located must communicate with the lumen (confirmed by serial section). Note the external incomplete ring near media. Intimal hemorrhage is present. $\times 12$

In all cases the thrombi overlay atherosclerotic plaques. Table 26-5 indicates that intramural hemorrhage was found in the underlying atheroma in all but two instances. Frequently the superficial layers of the plaque immediately beneath the thrombus were necrotic. In this table, *inconsistency* means that the oldest part of the thrombus did not overlie the area of maxi-

mal hemorrhage, and in seven cases the oldest area of thrombus was not associated with the area of maximal intimal hemorrhage. In three cases there was an association between thrombus and hemorrhage. Rupture of the atheromatous plaque was

TABLE 26-5 RECENT CORONARY THROMBOSIS

Number of cases	Intramural hemorrhage	Necrosis of plaque	Inconsistency of plaque	Rupture of plaque
14	12	8	7	4

found four times, and was always associated with massive hemorrhage in the atheroma. In serial sections, rupture usually occurred at more than one point. Our interpretation of the massive accompanying hemorrhage is that it derived from the lumen and was in the nature of dissection of the plaque (Figs 26-6, 26-7, and 26-8).

Discussion

Although intimal blood vessels have been recognized for many years,³ the work of



FIG 26-5. Endothelium-lined channels (central, large) lying superficially in the intima. Subsequent sections showed a plexus communicating with the lumen. $\times 240$.

The most convincing proof of the encrustation theory is provided by Duguid's illustrations, which show crescentic thickenings, often multiple, in coronary arteries, the most superficial and recent clearly being thrombus in an early stage of organization. Duguid regards the lipid pool as a late degenerative change, associated especially with red thrombi, and due to retarded organization. Geiringer supports Duguid and considers the softened center of a plaque as the result of necrosis owing to failure of organization.

I am in agreement with the above views and believe that atheromata with pools of lipid and marginal vascularization are derived from mural thrombi containing large admixtures of red blood cells in which degeneration proceeds at a faster rate than organization by endothelium. The granulomatous inflammation frequently found about the fatty material would represent the reaction to dead tissue. The small hemorrhages and admixtures of red blood cells, so often seen in our experience in these athero-



FIG 26-12 The most distal section of thrombus, showing early recanalization, a thick hyaline plaque with a few irregular spaces containing hemorrhage



FIG 26-11 A more distal section showing very marked distortion of lumen now at the upper left, containing thrombus but with loss of continuity of intima and some admixture of cholesterol and thrombus

phous pools of lipid, may then be the leakage of cells seen about newly formed and proliferating capillaries, as described by Sanders and French¹⁸ in healing wounds and by Waters¹⁹ in the organization of lipid injected into the cornea of the rabbit. Seepage of blood into such areas would add to the volume and may be a factor in growth and progression of the lesion. Larger intimal hemorrhages may be related to anoxia but our evidence for this, as stated earlier, is only suggestive. One also has the distinct impression that large hemorrhages, with many foam cells and delicate endothelial-lined channels traversing the superficial areas and little or no overlying hyaline cap, represent early organization of recent mural thrombi (Figs 26-9, 26-10, 26-11, and 26-12).

There is no doubt that channels of luminal origin nourish the superficial layers of the thickened intima and it is possible that occlusion of these vessels may result in infarction of the plaque and leakage of red cells into the necrotic center. Geiringer supports this view and speaks of hemorrhagic infarction of the plaque.

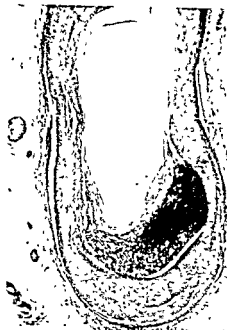


FIG 26-13 Nonoccluding thrombus, organizing, simulating atheroma with hemorrhage. Serial section of the entire segment proved the nature of the lesion. Note the hyalinization of surface layers $\times 12$

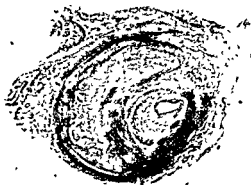


FIG 26-14 Pale inner vascular zone suggests a thrombotic origin although classically this is atheromatous narrowing $\times 9$

Despite the hemodynamic concepts of Paterson¹⁸ and Texon,²⁰ I am reluctant to accept the belief that pressures within intimal capillaries of a magnitude sufficiently greater than intraluminal pressure can cause hemorrhage into atheromatous plaques.

The variable morphologic appearance of atheroma may be related to the composition

of the original mural thrombus. For example, the hyaline, laminated eccentric type may be derived from encrustations consisting principally of fibrin and platelets (Fig 26-13). The presence or absence of ultimate calcification would be then determined by the speed of organization, and by the adequacy and integrity of newly formed intimal channels and, in this respect, would be similar in concept to the amorphous pools of lipid and necrotic material (Figs. 26-14 and 26-15).

One can only speculate about the possible reasons for recurrent thrombosis. In our material we did not consider intramural hemorrhage a convincing mechanism. As mentioned earlier, there was necrosis of the plaque beneath the thrombus. This we felt could be due to occlusion of luminal blood vessels but, whether this came first and led to the development of the overlying thrombus or, conversely, was the result of the thrombus occluding luminal vessels, we could not determine. It is also possible that the cause of the thrombosis remains the same for subsequent episodes as it was for the first and may be related to changes in endothelium associated with altered hemodynamics and increased coagulability of the blood. The exception to this concept would be the

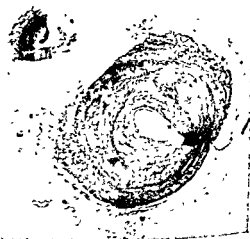


FIG. 26-15. The most distal section of thrombus, showing early recanalization, a thick hyaline plaque with a few irregular spaces containing hemorrhage. $\times 9$.

cases of massive hemorrhage described above. Here it is our conception that tears in the delicate overlying endothelium permitted dissection of a plaque by blood from the lumen.

Summary and Conclusions

1 The coronary arteries from 100 unselected adult autopsies were examined by perfusion and clearing, and atherosclerosis Grade III and Grade IV was encountered in 63 cases.

2 Intramural hemorrhage of recent origin was found in 51 cases and was always associated with amorphous pools of lipid showing marginal vascularization.

3 In at least 50 per cent of these cases there were intimal blood channels derived from the lumen.

4 Recent thrombosis was encountered 14 times and was associated with intramural hemorrhage in 12 instances, although the oldest part of the thrombus did not correspond with the area of maximum hemorrhage in seven cases.

5 Massive hemorrhage into an atheromatous plaque was associated with four thrombi. It is considered that the primary event in these cases is dissection of blood from the lumen into a soft and necrotic atheroma.

6 Evidence in support of the Duguid-Rokitansky hypothesis, that atheromata derive from mural thrombi, is discussed.

7 It is considered that the following mechanisms may come into play in the production of intramural hemorrhages.

a They may be due to leakage of blood from delicate organizing marginal capillaries when the hemorrhage is small.

b They may be associated with local anoxia when the hemorrhage is large.

c They may be associated with necrosis of the plaque which, in turn, may be due to interference with the luminal blood vessels.

d They may not, at times, be hemorrhage at all, but may represent incompletely organized mural thrombi containing many red blood cells.

8 Intimal hemorrhage is not considered

to be a cause of thrombosis of the lumen of the artery.

BIBLIOGRAPHY

- 1 DURLACHER, S. H., FISK, A. J., and FISHER, R. S. Lesions of coronary arteries, improved technique for their demonstration, *Lab Invest* 2:261, 1953.
- 2 YATER, W. M., WELSH, P. P., STAPLETON, J. F., and CLARK, M. L. Comparison of clinical and pathologic aspects of coronary artery disease in men of various age groups, study of 950 autopsied cases from Armed Forces Institute of Pathology, *Ann Int Med* 34:352, 1951.
- 3 WOODRUFF, C. E. Vasa vasorum, *Am J Path* 2:567, 1926.
- 4a PATERSON, J. C. Vascularization and hemorrhage of intima of arteriosclerotic coronary arteries, *Arch Path* 22:313, 1936.
- 4b PATERSON, J. C. Relation of physical exertion and emotion to precipitation of coronary thrombi, *JAMA* 112:895, 1939.
- 5 WINTERITZ, M. C., THOMAS, R. M., and LeCOMPTE, P. M. *The Biology of Arteriosclerosis*. Springfield, Ill., Charles C. Thomas, 1938.
- 6 WARTMAN, W. B. Occlusion of coronary arteries by hemorrhage into their walls, *Am Heart J* 15:459, 1938.
- 7 GEHRINGER, E. Intimal vascularization and atherosclerosis, *J Path & Bact* 63:201, 1951.
- 8 DUGUID, J. B. Mural thrombosis in arteries, *Brit Med Bull* 11:36, 1955.
- 9 ROKITANSKY, CARL. *A Manual of Pathological Anatomy*. Trans. by G. E. Day. Philadelphia, Blanchard & Lea, 1855, vol. 4, p. 205.
- 10 HEUKING, E., and THOMA, R. Ueber die Substitution des marantischen Thrombus durch Bindegewebe, *Arch f path Anat* 109:228, 1887.
- 11 McLELLINE, N. G. B. Pathogenesis of atheroma, *Am J Path* 28:413, 1952.
- 12 LICHTENBERGER, E. Quoted by J. B. DUGUID, *Brit Med Bull* 11:36, 1955.
- 13a LEVENE, C. I. The electron-microscopy of atheroma, *Lancet* 2:1216, 1955.
- 13b LEVENE, C. I. The early lesions of atheroma in the coronary arteries, *J Path & Bact* 72:79, 1956.
- 13c LEVENE, C. I. The pathogenesis of ather-

- oma of the coronary arteries, *J Path. & Bact.* 72:83, 1956.
14. VAN DEN HOOFF, A. Electron-microscopy of atheroma, *Lancet* 2 1392, 1955.
15. FLOREY, H W: "Some Problems Associated with Atherosclerosis" Lecture delivered at the University of Toronto, June, 1956. *Not published*
16. SINAPIUS, D. Ueber das Aortenendithel, *Arch. path Anat* 322 622, 1952.
17. SHORT, R H D Orientation of structure and of thrombi in the deep veins of the leg in man, *J. Path. & Bact.* 68:41, 1954
18. FRENCH, J. E., and SANDERS, A G Quoted by FLOREY¹⁵ *Unpublished data*
19. WATERS, L L.: Effect of vascularization on lipid-connective tissue reactions in the cornea, *Circulation* 14 487, 1956
20. TEXON, M.: A hemodynamic concept of atherosclerosis, with particular reference to coronary occlusion, *A M A, Arch. Int Med* 99:418, 1957.

CHAPTER 27 THE CAUSAL RELATIONSHIP OF STRESS TO INTIMAL HAEMORRHAGE AND CORONARY OCCLUSION

J. C. Paterson

Twenty years have passed since the hypothesis was advanced that intimal haemorrhage was intimately concerned in the production of coronary occlusion, either by secondary thrombosis^{1,2} or by massive intimal haematoma^{2,3}. This hypothesis was built up from four interdependent observations and deductions (1) that intimal haemorrhages are usually associated with coronary thrombi, (2) that most coronary thrombi (or occlusions by haematomas) are initiated by intimal haemorrhages; (3) that intimal haemorrhages are the result of rupture of intimal, *high-pressure* capillaries, and (4) that increased intracapillary pressure due to stress may be a factor in the rupture of intimal capillaries.

The evidence for and against these observations and deductions that has accumulated during twenty years will be given in the present paper, with the emphasis of course on the defence.

THE INCIDENCE OF INTIMAL HAEMORRHAGE WITH CORONARY THROMBI

Ever since a relationship between intimal haemorrhage and coronary thrombosis was first suggested,¹ a wide variation in the frequency of this relationship has been reported by different workers. In my own original series, in which 58 recent thrombi were studied by serial sections at short intervals, intimal haemorrhages were found in association with the thrombi in 89 per cent of the cases.⁴ But in my second series, in which the interval between serial sections was widened appreciably, the incidence of haemorrhage fell to approximately 62 per cent.⁵ Other workers have reported a similar reduced incidence with the technique of interrupted serial section, viz: Horn and Finkelstein,⁶ 64 haemorrhages in 123 cases (59 per cent), Wartman,⁷ 44 haemorrhages in 77 cases (58 per cent); Drury,⁸ 16 haemorrhages in 26 cases (62 per cent).

- oma of the coronary arteries, *J. Path. & Bact.* 72 83, 1956.
14. VAN DEN HOOFF, A. Electron-microscopy of atheroma, *Lancet* 2 1392, 1955.
 15. FLOREY, H. W.: "Some Problems Associated with Atherosclerosis" Lecture delivered at the University of Toronto, June, 1956 *Not published*
 16. SINAPIUS, D. Über das Aortenendithel, *Arch. path. Anat.* 322 622, 1952.
 17. SHORT, R. H. D. Orientation of structure and of thrombi in the deep veins of the leg in man, *J. Path. & Bact.* 68 41, 1954.
 18. FRENCH, J. E., and SANDERS, A. G. Quoted by FLOREY.¹⁵ *Unpublished data.*
 19. WATERS, L. L.: Effect of vascularization on lipid-connective tissue reactions in the cornea, *Circulation* 14 487, 1956.
 20. TEXON, M.: A hemodynamic concept of atherosclerosis, with particular reference to coronary occlusion, *A.M.A. Arch. Int. Med.* 99.418, 1957.

cells into the adjacent tissues. The second and better argument is that intimal haemorrhage, in general, cannot be due to thrombosis since it occurs so often in atherosclerotic plaques that have no associated thrombosis. One might postulate, of course, that a different mechanism exists for intimal haemorrhage with thrombosis than for haemorrhage without thrombosis; but this would appear unreasonable.

The fact that all intimal haemorrhages do not produce secondary thrombosis has been used as an argument against their thrombogenic properties. This argument cannot be defended on general pathologic principles. Everyone will agree that the primary pulmonary complex in children does not always result in fulminating tuberculosis. Nor does chronic retention of urine in adults always lead to acute pyelonephritis. Many other examples might be given, but it is well recognized that additional factors are often required for the production of sequelae to primary lesions. Applying this to intimal haemorrhage, it would appear that additional factors are also required here. Some of these, like the raw surface produced by an atheromatous ulcer from extrusion of the haemorrhage, will be mentioned later.

By a process of elimination, then, one can deduce that intimal haemorrhage, or lesions secondary thereto, have a causal relationship to most coronary thrombi. The microscopic evidence supports this deduction. If a thrombus occupies only part of the arterial lumen (as it did in two cases in my original series²), it will be found to be attached on that aspect of the vessel wall into which haemorrhage had occurred.

The mechanism by which intimal haemorrhages initiate thrombosis deserves consideration. By their very nature these haemorrhages are disruptive lesions, and it may therefore be assumed that thromboplastic substances are liberated from the injured area and make their way by diffusion to the endothelial surface of the vessel. If conditions of blood flow are suitable, thrombotic materials will be deposited at that point. Another way that haemorrhages may initiate

thrombosis is the result of their space-occupying properties. The tension within the haemorrhagic area may be so great that rupture of the plaque occurs, with extrusion of its haemorrhagic contents into the lumen, and the raw surface so produced may be the initiating factor in thrombosis. Rupture of the plaque will also be facilitated, no doubt, by interference with the nutrition of the intima from the break in its capillary circulation. And finally, retrograde capillary thrombosis may occur at the point of capillary rupture, and when the thrombus reaches the origin of the capillary it may initiate a similar process in the lumen itself.¹³

Mention should also be made of the possibility that intimal haemorrhages might occlude the arterial lumen by virtue of their bulk alone, without secondary thrombus deposition. Wartman has stressed this mechanism and believes that it occurs in approximately 10 per cent of cases of acute coronary occlusion.³ My own series yields similar results, although I have not been as impressed as Wartman with the mechanism in question. My doubts arise from the fact that the calibre of the arterial lumen prior to the development of haemorrhage cannot be measured, and the stenosing effect of intimal haemorrhage may therefore be more apparent than real.² Nevertheless, the addition of blood to an atherosclerotic plaque must increase its bulk; this increase in bulk should be reflected inward because of the restraint offered by the dense adventitial tissues,¹⁴ and so, if the haemorrhage is large, a significant degree of stenosis of the arterial lumen might theoretically occur.

Another argument against the occluding properties of intimal haematomas has arisen since routine anticoagulant therapy for coronary occlusion began. It has been argued that intimal haemorrhages of large size, and occlusive phenomena therefrom, should be encountered commonly in patients receiving anticoagulant therapy. However, it is a matter of record that occlusive phenomena in patients of this type are uncommon. The explanation for this inconsistency would appear to be that the production of a massive

intimal haematoma depends on factors other than blood coagulability. My own experience has been that haemorrhages of the coronary arteries in patients who had received dicumarol therapy were no larger than haemorrhages in patients who had not received it.¹² As will be seen in the last section of this paper, the factors that favour progressive bleeding into plaques are probably related more to pressure differentials in the arterial lumen than to blood coagulability.

THE CAPILLARY ORIGIN OF INTIMAL HAEMORRHAGE

Haemorrhagic lesions within atherosclerotic plaques have been noted for many years, but only recently has their mechanism been understood. At one time they were considered to be due to the reflux of blood from the lumen into the intima through a defect produced by atheromatous ulceration.¹⁴ However, while working with the late Professor Oskar Klotz during 1935-37, I was able to demonstrate an intact layer of intimal tissue between the haemorrhage and the lumen of the artery on three separate occasions.¹² Because of these very definite findings, intimal haemorrhages must, in my opinion, be regarded as intrinsic lesions, and when a defect in the intima is noted, it should be interpreted as a blow-in into the lumen, not a blow-out from the lumen (see next section for the physical principles involved).

The cause of the haemorrhage must therefore be sought within the substance of the sclerotic intima, and here the presence of capillaries in the immediate vicinity of haemorrhage offers an obvious explanation for the extravasated blood. These capillaries have been found to arise from the main arterial lumen on many occasions,^{1,7,9,11,12,14,15,16} hence, they have been referred to as *high-pressure* capillaries. They anastomose on occasion with the usual *vasa vasorum*,¹⁶ but in my experience this is an end-stage effect and is not present in early plaques. In any event, capillaries can always be found in intimate

relationship to the haemorrhages, and there was general agreement, up to 1951, that the extravasated blood was derived from their rupture. This obvious mechanism has been challenged recently by Drury.⁸ He has been so impressed with the frequent location of haemorrhages in the subendothelial tissues of the intima that he considers them to be the result of increased permeability or tearing of the endothelium, and not of capillary origin. I do not find this explanation convincing. Many intimal haemorrhages are deep-seated, not subendothelial, and complete serial section of these has failed to reveal anything in the nature of a tear or laceration of the intima. So why should the cause of the superficial ones be different from that of the deep-seated ones?

THE EFFECT OF STRESS ON THE INTEGRITY OF INTIMAL CAPILLARIES

Evidence has been presented in the preceding sections that intimal haemorrhages are associated commonly with coronary thrombi, that they are the usual precipitating lesions in thrombus deposition, and that they are caused by the rupture of capillaries which arise from the lumen of the main artery. The present section will deal with the proposition that the temporary elevation in blood pressure and the increase in coronary blood flow that result from physical or emotional stress may be factors in the rupture of these specialized capillaries. If this can be proved, it follows that stress may be concerned in the production of coronary occlusion, either by an haematoma *per se* or by secondary thrombus precipitation. This is the proposition that has a direct relationship to Workmen's Compensation Board cases, it is also the one that has attracted the widest attention and most of the disagreement.

My own views on the subject will be reviewed first. The crux of the matter to me is the peculiar origin and location of intimal capillaries. They arise directly from the lumen of a large vessel in which the pressure of blood, normally high, will be reflected

directly into the capillary lumen. With hypertension, either of the persistent variety or transitory from stress, the intracapillary pressure will be elevated even further, thus increasing the tendency to rupture^{1,2,17}

It is unfortunate that these propositions cannot be subjected to experimental proof, but the fact is that atherosclerotic plaques in experimental animals are either not vascularized or, if they are, they do not show intimal haemorrhages or coronary thrombi. The problem, therefore, can only be solved by deduction, and I will give first some of the objections to the simple hypothesis just described.

Master and associates have presented clinical evidence that stress is not related to the rupture of intimal capillaries.¹⁸ They found that the onset of the syndrome of coronary occlusion occurs in the majority of cases while the individual is either asleep or at rest. Only 2 per cent of cases in their series were associated with unusual exertion. From these facts, they conclude that intimal haemorrhage (which they admit is the usual first step in the process of occlusion) is independent of external factors, that it is probably "a fortuitous event." Their argument implies that coronary thrombi are initiated and progress to the point of occlusion in a short space of time—but for this there is no pathologic basis. Complete serial sections of recently occluded vessels from cases of sudden death without myocardial infarction have revealed that most coronary thrombi are built up gradually, that their oldest portions may be many hours or days in age as shown by the extent of organization.^{18,17} Thus, the initiating factor for the original thrombus deposition (*viz.*, capillary rupture) must be sought not at the time of clinical occlusion but in the hours or days preceding it.

King has presented biophysical evidence in an attempt to break down the hypothesis that intimal haemorrhage is concerned in the mechanism of coronary occlusion, with or without secondary thrombosis.¹⁹ He states in his conclusions that intimal haemorrhages "have been given an importance and signifi-

cance out of all proportion to their real status. It is necessary to look elsewhere in the physiology of the circulation for the explanation of cardiac infarction in these cases. It is not due to vascular obstruction from subintimal haemorrhages of coronary vessels." These conclusions of King's are surprising in view of the biophysical measurements given in the body of his paper. Some of these measurements will be used against him in the succeeding paragraphs.

I will begin with the basic assumption that intimal haemorrhages are intrinsic lesions, and that they are due to the rupture of capillaries that are derived from the lumen of the artery, from the *vasa vasorum*, or from both.

The addition of blood to an atherosclerotic plaque must increase its bulk. Intimal haemorrhages are thus space-occupying lesions, and the displacement should occur inward because of the restraining influence of the great tensile strength of the adventitia.¹⁶ Inward displacement must, therefore, mean that the pressure within the haemorrhagic area is greater than the pressure in the adjacent arterial lumen. Since the haemorrhage is due to capillary rupture, the intracapillary pressure at the point of capillary rupture must also be greater than the pressure within the adjacent arterial lumen. In this connection, the frequent demonstration, histologically, of superficial intimal capillaries that are ballooned and crowded with red cells suggests that the pressure within them during life was greater than that in the adjacent arterial lumen, otherwise they would be collapsed and bloodless.²¹ We must search, therefore, for circumstances in which the pressure in intimal capillaries is greater than the pressure within the lumen of the artery, at any one phase of the cardiac cycle, *e.g.*, diastole.

At this point it will be profitable to apply Bernoulli's principle to the problem. As stated by King,¹⁹ this principle depends on the fact that "when fluid passes through a constriction its speed (and therefore its kinetic energy) is increased, while its pressure energy is decreased. If the lumen is narrowed

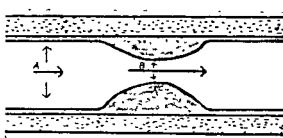


FIG 27-1 Diagram of a coronary artery showing a point of stenosis of the lumen by an atherosclerotic plaque. The lateral pressure at B will be lower than that at A during rest, and much lower during exercise

sufficiently, the kinetic energy may be so raised that the pressure energy may be very low in this region. This phenomenon is seen and utilized in the everyday world in the water suction pump seen in the laboratory, in the Venturi tube used in gasoline carburetors and in jet engines." King has adapted this principle to a stenosing lesion in a major coronary artery (Fig 27-1), and his calculations of the differences in lateral pressure developed at two specified points in the artery are summarized in Table 27-1.¹⁹ It will be seen from this table that the lateral pressure at the point of stenosis (B) is

TABLE 27-1 VARIATIONS IN LATERAL PRESSURE IN ATHEROSCLEROTIC CORONARY ARTERIES*

Diameter of arterial lumen at widest part (A)	Area of arterial lumen at point of stenosis (B)	Drop in lateral pressure at point of stenosis (B)	
		Mm H ₂ O	Mm Hg

BLOOD FLOW IN A MAJOR CORONARY ARTERY AT

REST (250 $\frac{1}{4}$ C CM / MIN) †

3 mm	$\frac{1}{2}$ that at A	3.5	<1
3 mm	$\frac{1}{3}$ that at A	9.4	<1
3 mm	$\frac{1}{4}$ that at A	17.6	1.3
3 mm	$\frac{1}{6}$ that at A	41.2	3.0

BLOOD FLOW IN THE SAME MAJOR CORONARY ARTERY DURING EXERCISE (1500 $\frac{1}{4}$ C CM / MIN) ‡

3 mm	$\frac{1}{2}$ that at A	126	9.3
3 mm	$\frac{1}{3}$ that at A	338	24.9
3 mm	$\frac{1}{4}$ that at A	634	46.8
3 mm	$\frac{1}{6}$ that at A	1023	75.2

* After E. S. J. King, *Australasian Ann Med* 18, 1952

† King estimates the flow in a first division of one coronary artery as a quarter that of total coronary flow.

‡ Quoted by King from Lovatt Evans, 1949

always less than that at the dilated part of the artery (A) proximal to the stenosis. When the individual is at rest and blood flow is minimal (250 c cm/min) the decrease in lateral pressure at the point of stenosis is slight; but it is great during conditions of exercise when blood flow rises to 1500 c.cm./min. These latter conditions are the ones that interest us in this Symposium on Work and the Heart. In King's own words, "it is thus easy to see that it is at times of increased blood flow that haemorrhage into the wall of the vessel is likely to occur. . . ." It is also apparent from Table 27-1 and Figure 27-1 that a stenosing plaque which has been enlarged by a space-occupying haemorrhage will cause even greater stenosis of the arterial lumen, an additional fall in lateral pressure at this point will result, and there should be enhancement of the tendency to capillary bleeding. A plausible explanation by physical principles for occlusion of the arterial lumen by intimal haematoma per se can therefore be given. Indeed it is difficult to understand why intimal haemorrhages do not all become massive in these circumstances. It would seem that the restraint offered by tissue tension is all that keeps them small.

Returning now to the capillaries that might be responsible for intimal haemorrhage, four general types can be considered. These are illustrated in Figure 27-2: Type 1 capillaries are those that arise from the

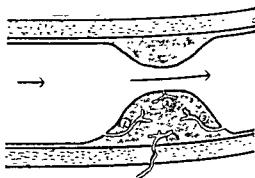


FIG. 27-2 Diagram of an atherosclerotic coronary artery showing four possible points of origin of intimal capillaries as described in the text

arterial lumen proximal to a point of atherosclerotic stenosis; Type 2, those that arise from the arterial lumen at the apex of the stenosing plaque; Type 3, those that arise from the arterial lumen distal to the stenosing plaque; and Type 4, those that are derived from the vasa vasorum and penetrate the media to lie in the outer layers of the plaque. Which of these capillaries, then, can be exposed to a pressure greater than the pressure in the arterial lumen adjacent to a stenosing plaque?

Capillaries that are derived from the vasa vasorum (Type 4) can probably be eliminated from consideration since the pressure within them is relatively low, being like that of ordinary capillaries in other parts of the body. This pressure in ordinary capillaries is in the range of 15-30 mm Hg and it is not appreciably elevated in hypertension or in stress. Unless the atherosclerotic plaque is extraordinarily large, stenosing the arterial lumen to a pinpoint and thus producing an enormous drop in lateral pressure (Table 27-1), it is doubtful if the pressure differential needed for capillary rupture could be attained, even during violent exercise.

It can be stated categorically that Type 2 capillaries that arise from the arterial lumen at the apex of atherosclerotic plaques cannot conceivably rupture when the individual is either at rest or at exercise. The pressure in the arterial lumen at the point of stenosis is low, and it is extremely low during exercise. The intracapillary pressure will be correspondingly low, therefore this type of capillary cannot rupture.

A different situation is encountered with those capillaries that arise from the arterial lumen proximal to a stenosing plaque (Type 1) and ramify within the substance of the plaque. Because they arise from the arterial lumen at a point where lateral pressure is high, the pressure within them will be approximately the same as in the artery, particularly if they are blind capillaries. It will be in excess of that in the arterial lumen at the point of stenosis, and thus more than that in the tissues which surround the distal portions of the capillary. If coronary blood flow

is increased and coronary blood pressure elevated by stress, the intracapillary pressure will be greatly in excess of the tissue pressure and there will be imminent danger of capillary rupture and intimal haemorrhage.

Finally, those capillaries that arise from the arterial lumen distal to an atherosclerotic plaque (Type 3) will be almost as vulnerable to rupture as those of Type 1, although the arterial pressure at their point of origin will be slightly less than that in the proximal segment of the artery. In any event, this type of capillary will also be susceptible to rupture during conditions of excessive blood flow.

One last type of capillary might be mentioned here—a capillary that arises like the one described as Type 1 but traverses the body of the plaque and anastomoses with the capillary described as Type 3. Since blood flow through a capillary cannot be fast, the pressure within it will correspond to that of the main artery either at the point of capillary origin or at its point of exit. Thus, in circumstances of increased coronary blood flow, this type of capillary also will be susceptible to rupture.

With these considerations in mind, I find it difficult to understand King's rejection of the intimal haemorrhage hypothesis of coronary occlusion.¹⁹ It would appear that he has made the fundamental error, for a pathologist, of assuming that practically all intimal capillaries arise at the apices of stenosing atherosclerotic plaques, i.e., at points where lateral pressures are markedly reduced. However, he admits in the body of his report that "there are conditions in which the capillary pressure may be significantly different from the pressure in the wall, such as when they (the capillaries) communicate with vessels (capillaries) which arise from the lumen of the main artery at a wider point." In this regard Winternitz has shown by injection techniques that capillaries arise not infrequently at the periphery of plaques,¹⁰ and we have noted the same thing in gross surface preparations when the capillary supply to the plaque is outlined by the alkaline phosphatase technique.²⁰

It should not be inferred from the foregoing that the rupture of intimal capillaries is dependent entirely on the height of intracapillary pressure. Other factors no doubt play a part, and of these abnormal capillary fragility would seem to be particularly important. This abnormal fragility might result from aging, from dietary deficiency, or from hormonal imbalance, even perhaps from the general softening effect of too much inactivity. Nevertheless, some degree of positive intracapillary pressure must be a prerequisite of rupture. A rotten garden hose rarely breaks and leaks when the tap is turned off.

BIBLIOGRAPHY

1. PATERSON, J. C. Vascularization and hemorrhage of intima of arteriosclerotic arteries, *Arch Path* 22 313, 1936
2. PATERSON, J. C. Capillary rupture with intimal hemorrhage as a causative factor in coronary thrombosis, *Arch Path* 25 474, 1938
3. WARTMAN, W. B. Occlusion of the coronary arteries by hemorrhage into their walls, *Am Heart J* 15 459, 1938
4. PATERSON, J. C. Some factors in causation of intimal haemorrhages and in precipitation of coronary thrombi, *Canad M. A. J.* 44 114, 1941
5. PATERSON, J. C. "The Reaction of the Arterial Wall to Intramural Haemorrhage," in *Symposium on Atherosclerosis*, Washington, D. C., National Academy of Science-National Research Council, Publ 338, 1955, pp 66-67
6. HORN, H., and FINKELSTEIN, L. E. Arteriosclerosis of the coronary arteries and the mechanism of their occlusion, *Am Heart J* 19 655, 1940
7. WARTMAN, W. B. "Factors Concerned in Narrowing or Occlusion of Coronary Vessels," in F. R. MOULTON (Ed.), *Blood, Heart and Circulation* Publ No 13, American Association for the Advancement of Science Lancaster, Pa., Science Press, 1940, pp 122-132
8. DRURY, R. A. B.: The role of intimal haemorrhage in coronary occlusion, *J Path & Bact.* 67:207, 1954.
9. YATER, W. M., TRAUM, A. H., BROWN, W. G., FITZGERALD, R. P., GEISLER, M. A., and WILCOX, B. B.: Coronary artery disease in men 18 to 35 years of age, report of 866 cases; 450 with necropsy examinations, *Am Heart J.* 36 334, 481, 683, 1948
10. VIKO, L. E.: Medico-legal problems of the heart in relationship to injury, *Utah Bar Bull.*, March-April, 1953
11. HAMILTON, J. D., and MOWBRAY, J. H.: Significance of intramural hemorrhage in coronary arteriosclerosis, *Circulation* 14:426, 1956
12. HEIFERN, M., and WEINBERG, S.: Personal communication to the author.
13. PATERSON, J. C. "Coronary Artery Disease," in McMANUS, J. F. A.: *Progress in Fundamental Medicine* Philadelphia, Lea & Febiger, 1952, p 190.
14. LEARY, T.: Experimental atherosclerosis in the rabbit compared with human (coronary) atherosclerosis, *Arch. Path.* 17 453, 1934
15. ROBERTSON, H. F. Vascularization of the thoracic aorta, *Arch Path.* 8 881, 1929
16. WINTERITZ, M. C., THOMAS, R. M., and LE COMPTE, P.: *The Biology of Arteriosclerosis* Springfield, Ill., Charles C Thomas, 1938
17. PATERSON, J. C. Relation of physical exertion and emotion to the precipitation of coronary thrombi, *J. A. M. A.* 112 895, 1939
18. MASTER, A. M., DACK, S., and JAFFE, H. L.: Activities associated with the onset of acute coronary artery occlusion, *Am Heart J* 18 434, 1939
19. KING, E. S. J.: The haemodynamics of sub intimal haemorrhage, *Australasian Ann. Med* 1 18, 1952.
20. PATERSON, J. C., MILLS, J., and MOFFATT, T.: Unpublished observations

CHAPTER 28 THE PATHOLOGY OF THE CONDUCTION SYSTEM AS RELATED TO GENERAL CARDIAC PATHOLOGY

Maurice Lev

The term, the conduction system, as used here includes the sinoauricular node, the atrioventricular node and bundle of His, the bundle branches, and the peripheral Purkinje network. This system is only rarely, if ever, involved independently, its pathologic change being almost always related to that of the heart in general.^{1,2} Thus, the conduction system is involved in congenital heart disease, hypertensive and arteriosclerotic heart disease, rheumatic heart disease, syphilitic heart disease, acute and healed myocarditis, endocarditis and acute pericarditis, and tumors of the heart. The pathologic changes found are degeneration, necrosis, inflammation, fibrosis, and elastosis. No tumor formation of the muscular parenchyma of the conduction system is known to exist, and the sinoauricular and atrioventricular nodes and the atrioventricular bundle do not undergo hypertrophy and atrophy.^{1,3,4}

In congenital heart disease, the atrioventricular node, bundle, and bundle branches may be altered in patent foramen primum,^{1,5} ventricular septal defect with or without pulmonary stenosis,⁶ aneurysm of the pars membranacea, cor triloculare biventriculosum,^{7,8} cor biatriatum triloculare with or without transposition of the arterial trunks,^{1,7} transposition of the ventricles,³ tetralogy of Fallot, the Eisenmenger complex, hypoplasia of the aortic tract complexes, coarctation of the aorta, tricuspid atresia complexes, fibroelastosis, bicuspid aortic valve with sclerosis, Ebstein's disease,⁹ mitral stenosis and atresia,^{2,10} and subaortic stenosis. There may be abnormalities in position and relationship of various components of the conduction system, or abnormalities in continuity, or accessory bundles may accompany or replace the normal structures. Interruption in continuity is usually found between the atrioventricular

node and bundle, or within the bundle. Such interruption may be a congenital abnormality per se, or due to secondary fibrosis. Absent right or left bundle branches or their mirror-image displacement¹¹ have been rarely noted. These pathologic changes may be associated with congenital atrioventricular heart block, right or left bundle branch block, or the Wolff-Parkinson-White syndrome.* Abnormalities in position without discontinuity may produce no electrocardiographic abnormality.

In hypertensive and arteriosclerotic heart disease, the conduction system may be affected by ischemic changes^{1,2,12,13} (Fig 28-1), or by contiguity to adjacent diseased strictures. Where there is narrowing of the right coronary artery proximal to the origin of the ramus ostii cavae superioris, there may be changes in the sinoauricular and atrioventricular nodes, the atrioventricular bundle, and the posterior radiation of the left bundle branch. Where there is narrowing of the right coronary artery distal to the origin of the ramus ostii cavae superioris, and proximal to the origin of the ramus septi fibrosi, then there may be changes in the atrioventricular node and bundle and the posterior radiation of the left bundle branch. Where there is narrowing of the posterior descending coronary artery distal to the origin of the ramus septi fibrosi, there may be changes in the posterior radiation of the left bundle branch. Where there is narrowing of the anterior descending artery in its first two centimeters, there may be changes in the right bundle branch. Where there is diffuse coronary arteriosclerosis or ischemia of the small coronary arteries, there may be diffuse changes in both bundle branches.^{14,15} In all the above contingencies there are associated ischemic changes in the myocardium which, in most cases, dominate the scene. Rarely, the ramus septi fibrosi or the ramus ostii cavae superioris may be exclusively narrowed, resulting in localized ischemic changes in the atrioventricular node and bundle or the sinoauricular node.

A separate category of arteriosclerotic change in the conduction system occurs owing to the spread of a degenerative pathologic

process from the aortic and mitral valves.¹⁶ This is seen in calcification of the annulus of the aortic leaflet of the mitral valve and the upper part of the muscular ventricular septum, adjacent to the pars membranacea, and less commonly in sclerosis of the base of the aortic valve. Here, a calcific or fibrocalcific lesion compresses or replaces the atrioventricular bundle, its bifurcation, or the left bundle branch. This may produce complete or incomplete atrioventricular heart block, or complete or incomplete bundle branch block.

In rheumatic heart disease, the conduction system may be involved in the acute phase or in the healing or chronic phases of the disease.^{1,2,17} In acute rheumatic myocarditis, the atrioventricular node, bundle and bundle branches may be involved not only by the myocarditis but by inflammatory changes in the central fibrous body, the base of the mitral and aortic valves, and the pars membranacea. Likewise, rheumatic pericarditis may involve the sinoauricular node. The above changes may be correlated with excitatory or inhibitory electrocardiographic phenomena. Mild degrees of atrioventricular block are common, but complete heart block is rare. In the healed phases of rheumatic valvulitis of the mitral and aortic valves, scarring may involve the atrioventricular bundle or either bundle branch by contiguity. In addition, healed arteritis may produce ischemic changes, especially in the bundle branches. Thus, partial or complete atrioventricular block or bundle branch block may accompany chronic rheumatic heart disease.

In syphilitic heart disease,^{1,2} gummatous myocarditis may involve and destroy the atrioventricular node or bundle. The diffuse type of syphilitic myocarditis may likewise affect the conduction system. Syphilitic involvement of the aortic valve and of the pars membranacea may likewise affect the bundle. In addition, right ostial stenosis may add an ischemic factor to changes in the conduction system. Thus, there may be complete atrioventricular block, bundle branch block, or excitatory electrocardiographic abnormalities.

In acute myocarditis associated with diph-



FIG 28-1 *Top* Acute necrosis of the left bundle branch in infarction, arrows point to the left bundle branch Hematoxylin-eosin stain $\times 300$ *Center* Acute necrosis of the right bundle branch in infarction. Hematoxylin-eosin stain $\times 300$ *Bottom* Fibrosis of the right bundle branch in ischemia, arrows point to the right bundle branch Hematoxylin-eosin stain $\times 150$



FIG 28-2 *Top* Acute inflammation of the atrioventricular bundle and beginning of the left bundle branch in acute myocarditis; arrows point to the bundle. Hematoxylin-eosin stain. $\times 30$. *Bottom*. Higher power of illustration at left, showing various mononuclear cells. Hematoxylin-eosin stain. $\times 150$.

theria,^{1,2,18} scarlet fever,¹ influenza, pneumonia, rubella, rubeola, mumps, typhoid fever, typhus fever, or of an unknown etiology.^{1,2,12} the conduction system may show changes (Fig 28-2). The involvement of the system may be greater or less than that of the atrial and ventricular myocardium. It has not been proved that the conduction system can be involved exclusively. The degree of involvement, in general, increases as one proceeds from the sinoauricular node to the bundle branches. However, instances have been re-

ported in which the sinoauricular node or the atrioventricular node have been the only structures involved in the conduction system. The involvement of the conduction system may be associated with partial or complete heart block, or excitatory phenomena such as nodal or ventricular tachycardia. In diphtheria, severe heart block is more common than partial heart block. In myocarditis associated with other diseases, partial heart block is the rule and complete heart block rare. In acute or subacute bacterial endocarditis of

the aortic and mitral valves and adjacent septal endocarditis, the inflammatory process may involve the atrioventricular node, bundle, or left bundle branch.^{1,2,13} In pericarditis of any source, the sinoauricular and atrioventricular nodes may be involved. In the pericarditis and the myocarditis of uremia, the atrioventricular node and bundle are especially involved with not infrequent atrioventricular block. Rarely, as in diphtheria, healed myocarditis may eventually result in fibrotic changes in the atrioventricular node and bundle with resultant atrioventricular block, and healed endocarditis may produce similar changes in the node, bundle, left bundle branch, and rarely the right bundle branch.

Primary or secondary tumors of the myocardium may secondarily involve and destroy the atrioventricular node and bundle. Myxomas, angiomas, and sarcomas have been reported affecting the conduction system. There is a rare, primary, benign cocciethelioma of the atrioventricular node that destroys the node and the beginning of the bundle, producing atrioventricular block.¹⁰ In addition, fatty infiltration of the pre-atrioventricular nodal area may isolate the node from the atrial musculature, and fatty infiltration of the atrioventricular node and bundle may also be associated with atrioventricular block.^{1,2} The pathogenesis of the block in hemochromatosis is unknown.

The pathologic changes in the conduction system enumerated in the above diseases usually, of themselves, are not correlated with appreciable functional impairment of the heart,¹⁰ with the exception of some of the tachycardias. The importance of the pathologic changes lies more in their serving as an indicator in diagnosis and prognosis. Thus, the presence of an atrioventricular conduction disturbance in coronary disease usually indicates severe narrowing of the right coronary artery. The existence of complete left bundle branch block in known coronary disease denotes narrowing of both the right and left coronary arteries. On the other hand, the presence of complete right bundle branch block in known coronary disease implies high narrowing of the anterior descending coro-

nary artery. Likewise, from the standpoint of prognosis, the occurrence of partial or complete atrioventricular block in coronary disease or complete atrioventricular block in myocarditis indicates a more serious prognosis.

BIBLIOGRAPHY

1. MONCKEBERG, J. G. *Untersuchungen über das Atrioventrikulärbündel im menschlichen Herzen*. Jena, Gustav Fischer, 1908.
2. MAILLARD, I. *Les maladies organiques du faisceau de His-Tawara. Les syndromes coronaires. L'endocardite septale. L'infarctus septal* (Etude clinique et anatomique). Paris, Masson, 1931.
3. TAWARA, S. *Das Reizleitungssystem des Säugetierherzens. Eine anatomisch-histologische Studie über das Atrioventrikulärbündel und die Purkinjeschen Fasern*. Jena, Gustav Fischer, 1906.
4. KESTIS, A., and FLACK, M. W.: The auriculo-ventricular bundle of the human heart, *Lancet* 2:359, 1906.
5. YATER, W. M., BARRIER, C. W., and McNABB, P. E. Acquired heart block with Adams-Stokes attacks dependent upon a congenital anomaly (persistent ostium primum), report of a case with detailed histologic study, *Ann Int Med* 7:1263, 1934.
6. YATER, W. M., LYON, J. A., and McNABB, P. E. Congenital heart block, review and report of the second case of complete heart block studied by serial sections through the conduction system, *JAMA* 100:1831, 1933.
7. MONCKEBERG, J. G. "Die Missbildungen des Herzens" HENKE, I., and LUBARSCH, O. *Handbuch der speziellen pathologischen Anatomie und Histologie*. Berlin, Springer, 1924, vol. II, p. 1.
8. YATER, W. M., LEAMAN, W. G., and CORNELL, V. H. Congenital heart block, report of the third case of complete heart block studied by serial sections through the conduction system, *JAMA* 102:1060, 1934.
9. LEV, M., GIBSON, S., and MILLER, R. A.: Ebstein's disease with Wolff-Parkinson-White syndrome, report of a case with histopathologic study of possible conduction pathways, *Am Heart J* 49:724, 1955.
10. DONOSO, E., BRAUNWALD, E., JACK, S., and GRISHMAN, A. Congenital heart block, *Am J. Med* 20:869, 1956.
11. ASCHOFF, L. *Die Bedeutung des Reizlei-*

- tungssysteme für die Bestimmung der Herzfehler, *J. Tech. Methods* 17-93, 1937.
12. LEV, M., and UNGER, P. N.: Pathology of the conduction system in acquired heart disease: severe atrioventricular block, *A. M. A. Arch. Path.* 60:502, 1955
 13. ROSENTHAL, S. R.: Branch arborization and complete heart block, *Arch. Int. Med.* 50:730, 1932
 14. YATER, W. M.: Pathogenesis of bundle branch block, review of the literature; report of sixteen cases with necropsy and of six cases with detailed histologic study of the conduction system, *Arch. Int. Med.* 62:1, 1938
 15. YATER, W. M., CORNELL, V. H., and CLAYTOR, T.: Auriculoventricular heart block due to bilateral bundle-branch lesions: review of the literature and report of three cases with detailed histopathologic studies, *Arch. Int. Med.* 57:132, 1936
 16. YATER, W. M., and CORNELL, V. H.: Heart block due to calcareous lesions of the bundle of His; review and report of a case with detailed histopathologic study, *Ann. Int. Med.* 8:777, 1935.
 17. GROSS, L., and FRIED, B. M.: Lesions in the auriculoventricular conduction system occurring in rheumatic fever, *Am. J. Path.* 12:31, 1936
 18. MARVIN, H. M.: The effect of diphtheria on the cardiovascular system: the heart in laryngeal diphtheria, *Am. J. Dis. Child.* 29:433, 1925.
 19. MAHAUX, I.: *Les tumeurs et les polypes du coeur: Etude anatomo-clinique*. Paris, Masson, 1915.
 20. SMITH, W. C., WALKER, G. L., and AIT, H. L.: Cardiac output in heart disease, complete heart block, auricular fibrillation before and after restoration to normal rhythm, subacute rheumatic fever and chronic rheumatic valvular disease, *Arch. Int. Med.* 45:706, 1930.

CHAPTER 29 THE STIMULUS TO THE REVASCULARIZATION OF MUSCLE: AN EXPERIMENTAL INQUIRY*

*George M. Hass, Richard H. Andresen,
Clarence W. Monroe, Fay Squire,
and Dorothy A. Madden*

INTRODUCTION

It is taken for granted that Nature provides an optimal blood supply to the myocardium as the heart adapts to increasing demands of the systemic circulation during normal growth. It has been suggested that the myocardium serves as the principal stimulus to development of its own blood supply and that the pattern of vascularization is directed by activity of the myocardium. If this idea is correct, an inquiry with respect to myocardial vascularization should deal primarily with the influence of muscle cells and more particularly the properties of muscle cells that act as a stimulus to vascularization. The view may be taken that

these properties are those which are most active metabolically and hence the most likely to attract and maintain a rich blood flow.

When the muscle cell is at rest, its metabolism may be concerned with matters that are not directly related to its mechanical functions. One fraction of this resting metabolism is concerned with maintenance of viability of the cell. Another fraction is concerned with intrinsic mechanisms aimed at increasing the mass of the cell by reduplication or hypertrophy, or for decreasing the mass by atrophy or disintegration. Still another fraction is concerned with intracellular restitution of systems modified by maintenance of tone or tension. When the muscle cell is mechanically active in doing work by contraction, there is a metabolic activity that requires an expenditure of energy over and above that used in its resting metabolism.

* This investigation was supported from grants (H-1630, HTS-5161) from The National Heart Institute, The Muscular Dystrophy Associations of America, Incorporated, and the Otto S. A. Sprague Memorial Institute.

The first question to arise is whether resting metabolism or metabolism concerned with mechanical activity is of greater importance in stimulating vascularization. Experience with denervation, immobilization, or prolonged inactivity of skeletal muscle indicates that metabolism concerned with mechanical activity is more important. With progressive reduction of sufficient mechanical activity, there is a progressive reduction of the capacity of the muscle cell to develop tension and to do work. This may be apparent before there is any decrease in the dimensions of the muscle cell or the volume of its blood supply. In time, however, atrophy of the muscle cell and diminished vascularity become recognizable and these changes may proceed until they are profound and irreversible. There is no proof that the foregoing rules of response of skeletal muscle to inactivity apply to the myocardium because there are few comparable studies of cardiac muscle. Available evidence, however, supports the opinion that reduction of cardiac muscle to a state of mechanical inactivity would lead to reduction in its capacity to do work with subsequent atrophy of muscle cells and decrease of myocardial blood supply.

ADAPTIVE RESPONSES OF MUSCLE TO DEMANDS FOR INCREASED WORK

Similarities between responses of cardiac and skeletal muscle are more easily recognized when the daily demand for performance of mechanical work approaches or exceeds the maximum capacity of the muscle to meet the demand. Under these conditions the muscle cells tend to undergo adaptive changes, a few of which may be mentioned. One adaptation is in the nature of an increased efficiency. A second adaptation is an increase in the dimensions and at times the number of muscle cells. A third adaptation is an increase in the blood supply of the muscle. Though these adaptive responses are usually closely related, let us consider them separately.

A given amount of normal skeletal muscle with a given normal blood supply may have

in health or disease a wide range in its capacity to do work. This range varies from person to person and from day to day in the same person. Therefore, 10 gm of normal muscle in one person is not necessarily the same as 10 gm, of structurally similar muscle in another person. This is generally admitted in comparing the capacity of equal amounts of skeletal muscle to do work in different persons. It is less generally admitted in comparing differences in capacities of equal amounts of cardiac muscle in different persons. Under conditions of health the obvious differences are usually lightly dismissed as due to "natural" causes. Under conditions of disease the obvious differences are frequently attributed to myocardial "toxins," minute scars, tiny inflammatory lesions, minor valvular defects, or moderate coronary arteriosclerosis. These explanations seldom withstand close scrutiny. More often than not it must be concluded that the reasons for a difference in the capacity of two apparently similar masses of cardiac muscle to do work will remain obscure until mechanisms that maintain a supply of chemical energy in the muscle cell and the intracellular mechanisms that use this energy in the performance of work are understood. Certainly, the mere provision of equal blood supplies to equal volumes of muscle does not assure provision of equal or adequate operation of these intracellular mechanisms, however strongly the mechanisms may serve as a stimulus to development of their own blood supply.

When a muscle cell is called upon to do more daily work than it has been accustomed to, it may do so without an increase in its mass. The means by which the cell carries out this adaptation is unknown. In general, however, when the normal daily capacity of any given muscle mass to do work is exceeded, an increase in the mass of muscle cell structure, volume of its circulation, and mass of its supporting connective tissue tends to occur. Temporal or quantitative relation between these changes is not clear but this response to a real or impending insufficiency of muscle is evident up to a curiously variable limit. The usual stimulus to this hypertrophy

seems to be the increased demand on the muscle to do mechanical work. It is hardly proper to regard this solely as a "work hypertrophy," because hypertrophy may occur without any increase in the work load when the capacity of the existing muscle to carry on its normal work is decreased. In this instance hypertrophy of muscle cells may occur even though the demand for work is less than that to which the muscle cells were accustomed. It may also be apparent when the distribution of the normal work load changes, either as the result of a depletion in number of muscle cells, a rearrangement of muscle cells, or a disturbance in integration of contractile function. But whatever the situation leading to myocardial hypertrophy in terms

of units, bundles, or chambers happens to be, it is generally conceded that the hypertrophy is accompanied by an increase in the blood supply. This is especially conspicuous when massive generalized or local cardiac hypertrophy with comparable enlargement of coronary arteries occurs in young people. It is less readily recognizable when the cardiac hypertrophy has developed in the presence of severe coronary atheroarteriosclerosis in older people. In this connection evidence indicates that myocardial hypertrophy may be limited if a corresponding development of an increased blood supply is hampered by certain conditions, especially calcific atheroarteriosclerosis and its complications. This may be due not only to the limited capacity of

FIG 29-1 Medium-power photomicrograph of the essential structure of the musculofascial grafts used in these experiments. The muscle fascia is indicated by *F*, the musculofascial zone by *MFZ*, and the skeletal muscle by *M*.



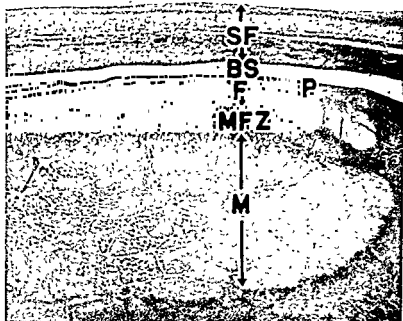


FIG 29-2 Low-power photomicrograph of a musculo-fascial autograft, 2 weeks of age. The subcutaneous fascia of the host is indicated by *SF*, the bursal space formed over the graft by *BS*, the fascia of the graft by *F*, the pannus which has grown over the fascia by *P*, the musculo-fascial zone by *MFZ*, and the residual muscle of the graft by *M*. The bed of the graft is composed principally of skeletal muscle of the host.

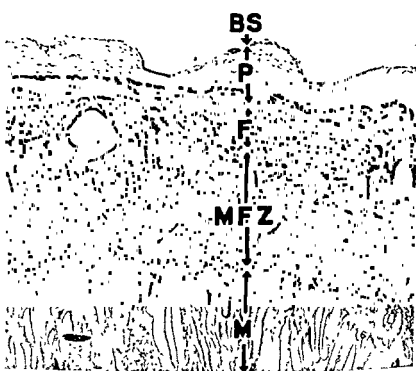


FIG 29-3 Medium-power photomicrograph of a part of an autograft, 2 weeks of age. The bursal space is indicated by *BS*, the pannus by *P*, the muscle fascia of the graft by *F*, the musculo-fascial zone by *MFZ* and the residual muscle of the graft by *M*. Note the rich vascularization of the pannus, the large vascular channels that extend from the pannus into the musculo-fascial zone, and the ample vascularizing stromal reaction with long regenerating strands of skeletal muscle in this zone.

diseased arterial channels to enlarge so as to accommodate demands of an increasing mass of muscle but also to other factors, not the least of which may be the limitation of intracellular mechanisms to create a demand for an increased blood supply.

The preceding comments offer some support to the idea that the maximum stimulus to vascularization and revascularization of the myocardium as well as development of ample collateral circulations is attributable principally to the demands of "viable" muscle cells working at a level close to that required to produce fatigue or hypertrophy. Unfortunately, the effectiveness of the stimuli to vascularization may be enhanced or retarded by many factors, only a few of which need be mentioned. The first of these is the availability of vascular channels from which a adequate supply of blood can be drawn. A second limiting factor is the capacity of the available vascular channels to undergo the required enlargement. A third is the entrenchment of atheroarteriosclerotic processes on the existing and newly established collateral channels. Nor can the more obscure but nevertheless important problem of modified diffusion gradients between capillaries and enlarging muscle cells be overlooked.

While admitting the limitations of adaptability of the blood supply as well as metabolic demands of muscle cells, it seemed that an experimental study of possible sources of stimuli to vascularization of muscle might be profitable. The experiments to be described at this time were designed to create conditions adverse to the survival of muscle and to assess the effect of these conditions on the development of a new circulation.

EXPERIMENTAL STUDIES

Normal Revascularization of Musculofascial Autografts

The first experiment, using New Zealand rabbits of the same source and strain, was done as follows. Blocks ($2.5 \times 2.5 \times 0.5$ cm) of erector spinae muscle with intact overlying muscle fascia were resected and implanted as autografts in locations comparable

to those from which the muscle had been excised.¹ These grafts were composed principally of a dense superficial layer of avascular collagenous fascia bound tightly to a broad subjacent layer of skeletal muscle at a narrow junction designated as a musculofascial zone (Fig 29-1). At the end of the first week, the transplants were firmly healed in place and a small bursal space had formed between the subcutaneous fascia and the

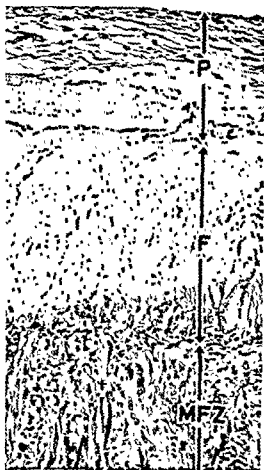


FIG 29-4 High-power photomicrograph of the active areas in an autograft, 2 weeks of age. The pannus between the bursal space and the muscle fascia is indicated by *P*, the muscle fascia by *F*, and the musculofascial zone by *MFZ*. Note the large vascular channel in the pannus, the perpendicular penetration of the fascia by vascular channels arising from those in the pannus, and the multinucleated abortive forms of regenerating skeletal muscle cells in the granulation tissue of the musculofascial zone.

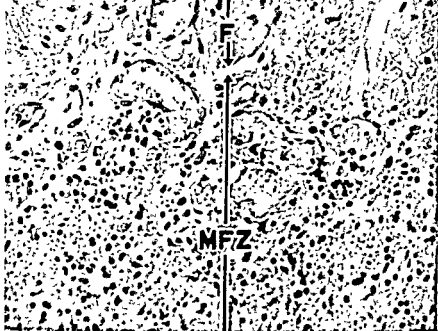


FIG. 29-5 High-power photomicrograph of the subfascial zone of a musculo-fascial homograft, 2 weeks of age. This was the third of a series of successive homografts from the same donor into the same recipient. The fascia of the graft is indicated by *F* and a part of the musculo-fascial zone by *MFZ*. Note the acute necrotizing hemorrhagic reaction and the thromboangiitic changes in the newly formed vascular channels subjacent to the fascia. Sterile acute reactions of this type interrupted the usual pattern of vascularization of homografts.

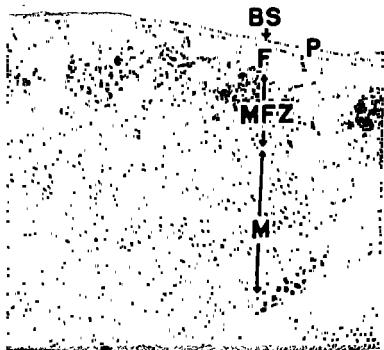
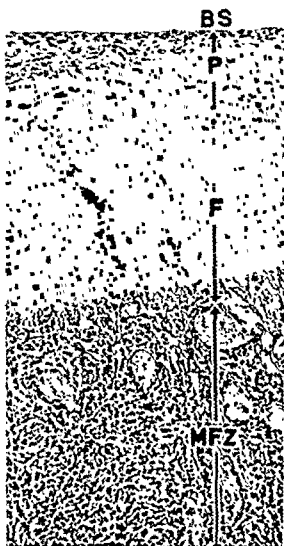


FIG. 29-6 Low-power photomicrograph of a homograft, 2 weeks of age. The bursal space is indicated by *BS*, the pannus by *P*, the muscle-fascia by *F*, the musculo-fascial zone by *MFZ*, and the muscle of the graft by *M*. This shows the diffuse infiltration of the pannus, fascia, and musculo-fascial zone by chronic inflammatory cells. This type of sterile chronic inflammation did not improve vascularization of the graft.

fascia of the graft (Fig. 29-2). The wall of the bursal space was composed of newly formed vascularized granulation tissue that resembled a pannus as it spread from the margins over the fascial surface of the graft. At the end of the second week, the pannus was almost as thick as the subjacent fascia. Enlarging vascular channels arising from tissues beyond the margins of the graft had fully permeated the pannus and penetrated the subjacent fascia perpendicularly to ramify throughout the musculofascial zone of the graft (Fig 29-3). As this occurred there were conspicuous changes in the musculofascial zone. Initially, it was a musculofascial junc-

tion without breadth but with the passage of time the muscle cells deteriorated and slowly disappeared. These events were accompanied by a conspicuous stromal reaction that filled in a broadening musculofascial zone, bounded above by the muscle fascia of the graft and below by the retreating margin of resorbing degenerate muscle of the graft. The stromal reaction was at its peak by the end of the second week and at this time abortive forms of regenerating skeletal muscle cells were abundant throughout the musculofascial zone (Fig 29-4). By the end of the third or fourth week the stimulus to the reaction had subsided. The original muscle of the graft had

FIG 29-7 Medium-power photomicrograph of a part of the homograft shown in Fig 29-6. The bursal space is indicated by BS, the pannus by P, the muscle fascia by F and the musculofascial zone by MFZ. Note the vascular channels in the pannus and in the musculofascial zone with communicating vessels passing through the fascia. The granulomatous sterile inflammation around these vessels and throughout the musculofascial zone was a deterrent to progressive vascularization of the graft.



deteriorated and resorbed. The pannus and musculofascial zone had become more densely collagenous. The rich capillary network had disappeared and the abortive forms of regenerating muscle cells were rapidly regressing. The sequences of degeneration, stromal proliferation, vascularization, abortive regeneration, and final repair were nearly completed.

The Effects of Acute Sterile Inflammation on Revascularization of Musculofascial Grafts

The second experiment was designed to determine the extent to which the pattern of vascularization of musculofascial grafts might be modified by the superposition of a sterile reproducible inflammatory reaction of an acute type. This was done by the use of successive homografts from the same donor to the same recipient.¹ It was shown that vascularization of the pannus, the muscle fascia, and the musculofascial zone followed the same sequences as those described in autografts for the first 5-7 days. At this time an acute thromboangiitic hemorrhagic reaction developed in the tissues involved by

the stromal reaction. This was particularly violent in the musculofascial zone during the second week (Fig. 29-5). The intense inflammation stopped the orderly sequences of stromal organization and failed to improve the effectiveness of stimulus to vascularization of degenerating muscle in the musculofascial zone.

The Effects of Chronic Sterile Inflammation on Revascularization of Musculofascial Grafts

The third experiment was designed to determine the effects of a reproducible, indolent, sterile, chronic inflammatory reaction on the pattern of vascularization of musculofascial grafts. This was done by the use of single musculofascial homografts.¹ Degeneration and resorption of muscle with vascularized stromal organization of the musculofascial zone began in much the same way as in the autografts. However, the completion of the process of stromal organization was interfered with by local appearance of host-homograft interactions during the second week. These interactions were characterized by progression of a chronic indolent type of inflamma-

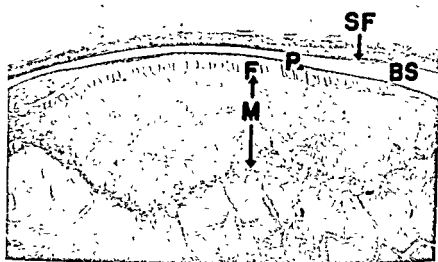
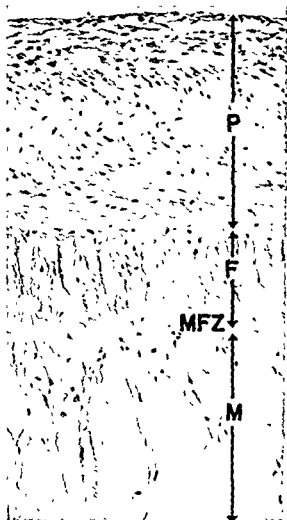


FIG 29-8 Low power photomicrograph of a homograft, 2 weeks of age, which was briefly exposed to a temperature of about -180°C . before implantation. The subcutaneous fascia of the host is indicated by SF, the bursal space by BS, the pannus by P, the muscle fascia of the graft by F, and the muscle of the graft by M. Though the homograft is well encapsulated by stromal reactions of the host, there is no vascularization. No resorption in the musculofascial zone has occurred. There is no inflammation. Compare this illustration with Fig. 29-6, which shows the customary host-homograft interaction.

FIG. 29-9 High-power photomicrograph of a field of the homograft shown in Fig. 29-8. The pannus is indicated by *P*, the muscle fascia by *F*, the musculofascial zone by *MFZ*, and the residual muscle of the graft by *M*. The pannus is avascular. There is no vascularization of the fascia. The musculofascial zone is nothing more than a junction. The muscle cells of the graft show no signs of atrophy, disintegration, or regeneration. These are the features of all autografts or homografts exposed to adverse low or high temperatures or to excessive x-irradiation *in vitro* before implantation in the recipient.



tion in the zones where vascularized stroma derived from the host was intimately admixed with proliferating tissues of the graft (Fig. 29-6). This reaction led to a reduction in the collagenous component of the stromal reaction, an increase in the reticuloendothelial, monocytic, and lymphocytic components and a decrease in orderly vascularization (Fig. 29-7). Meanwhile, the rate of degeneration and deterioration of muscle cells in the homograft remained essentially the same as that which occurred in control autografts. It was concluded that this type of chronic inflammation did not serve as an effective or useful stimulus to vascularization of degenerating muscle.

The Effects of Thermal Reduction of Viability of Musculofascial Grafts on Revascularization

The fourth experiment was designed to assess the relations between "viability" of cells of the graft and occurrence of a vascularizing stromal reaction. The previous studies with autografts and homografts had indicated that degeneration and perhaps abortive attempts at regeneration of muscle cells had a great deal to do with stimulating vascularizing stromal reactions of the host with subsequent organization and resorption of the graft. Although the muscle cells of the graft were initially deprived of a blood supply and were not fully "viable" by ordinary microscopic or functional standards, it

seemed clear that some intrinsic property of the cells served as a stimulus to vascularization. It was decided that further information about this property was needed. Hence, autografts and homografts were resected and subjected to a range of adverse temperatures *in vitro* before transplantation.² These studies showed that exposure of grafts for at least 20 minutes to temperatures below -20°C . or for at least 27 minutes at 56°C . eliminated all factors responsible for the characteristic autologous and homologous host-graft interactions. Instead, the autografts and homografts stimulated the same type of reaction in the host. This was characterized by negligible proliferation and delayed resorption of cells of the grafts. This tendency for the grafts to persist unchanged was accompanied by avascular encapsulation and indolent avascular penetration of each type of graft by stromal tissues of the host. In this connection, the following microscopic observations occurred (Figs 29-8 and 29-9). First, the bursal space over the muscle fascia of the graft formed promptly and a pannus regularly spread over the fascia. The usual

pannus, though rich in fibroblasts and collagen, was not supplied with capillaries. Second, the avascular stroma of the pannus did not penetrate the muscle fascia to the musculofascial zone of the graft. Third, the only stromal penetration and vascularization in the musculofascial zone were encountered at the extreme margins of the graft (Fig 29-8). Fourth, the muscle cells of the graft remained in apposition to the muscle fascia, showing minimal evidence of deteriorative resorption and no evidence of regeneration (Figs 29-8 and 29-9).

The temperatures at which the grafts lost the property of stimulating vascularization were not far removed from those believed to be lethal for most adult mammalian cells, though the term "lethal" has no adequate definition and we should not be content with its use.³ It might be better to conclude tentatively that the property of stimulating vascularization was lost by grafts previously exposed to thermal conditions that suppressed or destroyed the capacity of the cells of the graft to grow, divide, or undergo spontaneous atrophy.

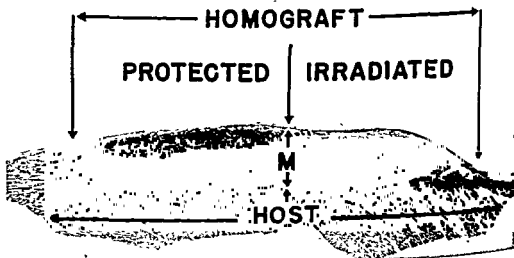


FIG. 29-10 Low-power photomicrograph of a homograft, 2 weeks of age. Prior to implantation, one half of this graft was exposed to 5,000 r of x-irradiation *in vitro* while the other half of the graft was protected from the irradiation with a lead shield. The part of the graft subjected to irradiation shows essentially the same features as the thermally treated graft illustrated in Figs 29-8 and 29-9. The part of the graft protected from irradiation shows the classical homograft-host interactions shown in Figs 29-6 and 29-7. Vascularization occurred only in the pannus, fascia, and musculofascial zone of the part of the graft, protected from x-irradiation.

The Effects of X-Irradiation Reduction of Viability of Musculofascial Grafts on Revascularization

The last experiment was concerned with another attempt to characterize the property of "viability" of grafts that seemed to promote vascularization by stimulating ingrowth of blood vessels from the graft bed. Autologous and homologous grafts were resected. They were then exposed *in vitro* to x-irradiation. The amount of irradiation given to a series of grafts varied from 90,000 r to 100 r. At times, one half of the graft was shielded with lead so that exact evaluation of the effect could be made by comparison of protected and irradiated halves of the same graft (Fig. 29-10). These studies showed that heavy x-irradiation (90,000-5,000 r) had about the same effect as excessive thermal exposure but after lesser amounts of radiation (1,000-100 r) gradations of effect essentially the same for autografts and homografts were recognizable. These gradations spanned a broad spectrum of increasing stimulation to vascularizing stromal reactions and customary host-graft interactions as the amount of irradiation decreased. It was concluded that the amount of x-irradiation known to be "lethal" or known to suppress the capacity of cells to grow and reproduce was close to the amount required to inactivate the property of musculofascial grafts that stimulates ingrowth of vascularized stroma from the bed of the graft.³ The delayed vascularization in many grafts after their exposure to low levels of irradiation indicated that inactivation of this property may at times be either incomplete, transient, or reversible.⁴

DISCUSSION

It may be of interest to speculate about relations between vascularization of musculofascial grafts under these experimental conditions and vascularization or revascularization of the myocardium under conditions of health and disease. In the first place, it may be assumed that the principal stimulus to myocardial vascularization depends upon

metabolic signals arising in the myocardium itself. The nature of these signals and the means by which they excite the sequences of vascularization and maintain an established blood supply are obscure. If we depend on clues presented by the experimental observations, we may conclude tentatively that these signals do not arise from muscle deprived of its ability to regenerate or actively degenerate. Furthermore, we may conclude that maintenance of an established blood supply depends upon a persistence of these signals. The means by which the signals reach the stromal receptors to excite the sequences and direct the pattern of vascularization is also a matter for speculation. The observations, however, indicate that the intensity of the stromal reaction depends on the intensity of the metabolic or bioelectric signals and that the direction of the vascularizing stromal reaction is along the shortest path perpendicular to the plane or locus of origin of the signals. This was shown well by the vascularization of the pannus as it spread out over the fascia of the graft and the perpendicular penetration of the fascia by newly formed capillaries aimed at the subfascial concentration of metabolic or catabolic activity of the muscle cells. The excessive vascularizing stromal reactions pointing toward the musculofascial zone also seemed to favor the view that the signals remained concentrated along a gradient that did not extend far from the site of their origin.

If significance is to be attached to these theoretical stimuli, it is proper to be concerned with their source. The source does not seem to be simply in the structure of muscle, for the persistent components of muscle "devitalized" by undue thermal exposure or x-irradiation did not serve as a stimulus to vascularization. Presumably the source was not simply in residual enzymatic activity of muscle, because many enzymatic functions of muscle cells and even the contractile capacities of intracellular myofibrils are retained in muscle subjected to "lethal" low temperatures.^{5,6,7} The source was dependent upon a still higher state of "viability" closely related, we believe, to the state of integration re-

quired for a natural biologic sequence of degradation or synthesis of cell structure that depends upon an adequate blood supply for its completion. We regard this particular state of "viability" as very close to the state required for occurrence of certain reversible changes in muscle related to the demand for work and the capacity of the muscle to do work. This leads to the idea that a lessening demand, a lessening mechanical activity, a lessening of metabolic signals, a degradation of structure, a transient increase in blood supply, and a subsequent reduction of circulation may go hand in hand. As a corollary, a steady demand, a lessened capacity to respond to the demand, a state of impending fatigue of the muscle cell, a generation of new structure with or without degeneration, an increase of metabolic signals, and an increase in circulation may go hand in hand. In any event the stimulus to development of an increased circulation would seem to be greater and more effective as the muscle approaches the limits of its capacity as manifest by the onset of fatigue, however contrary this might otherwise be to the best interest of the healthy or sick patient. Models of these situations, excepting those involving hypertrophy, are exemplified by the observed experimental sequences in the reactions in the modified and unmodified grafts.

SUMMARY

An experimental inquiry into the nature and source of stimuli required for vascularization and revascularization of muscle was made. A pattern of vascularized stromal penetration of skeletal musculofascial autografts was defined. A similar pattern initially appeared in homografts but the onset of sterile, acute, and chronic inflammatory sequences due to host-homograft interactions interfered with progressive vascularization. Exposure of grafts to adverse thermal conditions or sufficient x-irradiation in vitro before implantation eliminated the stimulus to vascularized stromal penetration of the grafts. These conditions were similar to those that destroy or suppress the capacity of muscle

cells to reduplicate themselves or to undergo spontaneous atrophy and degeneration but were not severe enough to destroy many other properties of components of "viable" muscle cells, such as those represented by activity of miscellaneous intracellular enzymes and myofibrillar contractile systems. The observations indicate that adequate vascularization or revascularization of cardiac muscle is not likely to occur unless stimuli arise from muscle cells that require a blood supply to support a level of "viability" at least equal to the level defined by these experiments with skeletal muscle. Further studies designed to inquire into the nature of these stimuli and the means by which they activate vascularizing sequences are needed.

BIBLIOGRAPHY

1. ANDRESEN, R. H., MONROE, C. W., and HASS, G. M. The pattern of tissue reactions to autologous and homologous musculofascial transplants, *J. Exper. Med.* 95:509, 1952.
2. ANDRESEN, R. H., MONROE, C. W., HASS, G. M., and MADDEN, D. A. Tissue reactions to autologous and homologous musculofascial transplants, modification by thermal treatment before transplantation, *A. M. A. Arch. Path.* 62:272, 1956.
3. REID, T. R., and GIFFORD, M. P. Quantitative study of effects of x-radiation on cells in vitro, *J. Nat. Cancer Inst.* 13:431, 1952.
4. FRIEDENWALD, J. S., BUSCHKE, W., and MOSES, S. G. Comparison of the effects of mustard, ultraviolet and x-irradiation, and colchicine on the cornea, *Bull. Johns Hopkins Hosp.* 82:312, 1948.
5. SCHICK, A. F., and HASS, G. M. The properties of mammalian myofibrils isolated by an enzymatic method, *J. Exper. Med.* 91:655, 1950.
6. ASHLEY, C. A., SCHICK, A. F., ARASMANIUS, A., and HASS, G. M. "Isolation and Characterization of Mammalian Striated Myofibrils," in *Connective Tissue*. New York, Trans. 4th Conference, Josiah Macy, Jr. Foundation, New York, 1953, pp. 47-141.
7. BROWN, A., ARAS, A., and HASS, G. M. Problems in standardization of isolated myofibril contractile systems in vitro, *Fed. Proc.* 15:509, 1956.

CHAPTER 30 INCIDENCE OF UNSUSPECTED HEALED MYOCARDIAL INFARCTION IN A GENERAL HOSPITAL

S. E. Gould and L. P. Cawley

How common is clinically unsuspected healed myocardial infarction among patients in a general hospital? In order to answer this question, we reviewed a series of 5,000 consecutive autopsies covering the years 1945 to 1955, inclusive, at this 500-bed general hospital. This is the county hospital of Wayne County in which the city of Detroit is located. In addition to the general hospital division, the institution has a psychiatric division with about 3,500 mental patients and an infirmary with about 2,500 indigent inmates (about 2,030 men and 470 women).

After selecting the autopsy protocols of cases with old myocardial infarcts, the corresponding clinical charts were reviewed. Notation was made of the age and sex of the patient, presenting complaints, history, and physical findings with particular reference to cardiac or vascular disease, occurrence of diabetes mellitus, and the electrocardiographic findings. All cases were ex-

cluded in which the clinical history, or electrocardiographic, radiologic, or physical findings led to a diagnosis or to suspicion of myocardial infarction, either old, recent, or fresh. A systolic blood pressure of 160 mm or over or a diastolic blood pressure of 100 mm or over was regarded as evidence of hypertension.

The autopsy protocols were reviewed with particular reference to presence of old or recent cerebral infarcts; severe narrowing, complete occlusion, or old or recent thrombi in either coronary artery or in any of the main branches of the coronary arteries; presence and location of old, recent, or fresh myocardial infarcts; ventricular aneurysm; and weight of the heart. Old infarcts were grossly white or gray white and microscopically were composed of fibrous or dense scar tissue. Localized isolated areas of fibrosis 0.5 cm or less in diameter were arbitrarily excluded. The heart of a man was regarded as hypertrophied if it weighed

400 gm. or more; and that of a woman, if it weighed 350 gm. or more.

RESULTS

MYOCARDIAL INFARCTS As may be seen in Table 30-1, myocardial infarcts (one or more) were found in 588 autopsies (11.76 per cent). In 136 autopsies (2.72 per cent) the infarct was fresh or recent and in 452 (9.04 per cent) old. Of the 452 hearts with old infarcts, in 277 (5.54 per cent), old, recent, or fresh infarction was diagnosed or suspected. In 175 autopsies (3.5 per cent) one or more old myocardial infarcts were found that were not clinically diagnosed or suspected.

TABLE 30-1 INCIDENCE OF MYOCARDIAL INFARCTS IN 5,000 CONSECUTIVE AUTOPSIES IN A COUNTY GENERAL HOSPITAL

<i>Infarcts</i>	<i>Number</i>	<i>Per cent</i>
Total number	588	11.76
Fresh or recent	136	2.72
Old diagnosed or suspected	277	5.54
Old unsuspected	175	3.5

Of the 175 hearts with old healed unsuspected infarcts, in 120 the infarct was located anteroseptally or laterally in the left ventricle, 48 posterobasally; six at the apex and base, and 1 in the right atrium. In addition, in 11 hearts with old apical infarcts, there was associated recent or fresh infarction and 1 heart with old infarcts both at the apex and base had a fresh apical infarct. Aneurysms were noted in 8 infarcts, 7 of which were apical.

PSYCHIATRIC STATUS Of the 841 psychiatric patients who came to autopsy, 12.1 per cent had infarcts (Table 30-2); of the 4,159 non-psychiatric patients, 11.7 per cent had infarcts.

RACE, AGE, AND SEX OF PATIENTS. The incidence of infarcts among white patients (12.8 per cent) was more than twice as great as that among Negro patients (6.0 per cent). Relatively more men than women were found to have infarcts (12.2 per cent of all men compared to 10.8 per cent of all

women). This disproportion between the sexes was slight for white patients (13.0 per cent of men compared to 12.2 per cent of women) but greater for Negroes (6.5 per cent of men compared to 5.0 per cent of women). The ratio of men to women with infarcts was 1.13 to 1.

The average ages of various groups of patients with infarcts of different types (fresh, old suspected, or old unsuspected) are given in Table 30-3. The average age of all 588 patients with infarcts was 68.2 years. The average age of all Negro patients with infarcts was 61.8 years, of those with fresh infarcts, 58.1 years, and of those with old suspected infarcts, 54.2 years. For every type of infarct, the average age of Negro women was less than that of Negro men.

TABLE 30-2 INCIDENCE OF MYOCARDIAL INFARCTION AMONG 5,000 CONSECUTIVE AUTOPSIES, ACCORDING TO PSYCHIATRIC STATUS, RACE, AND SEX

<i>Group of patients</i>	<i>Number</i>	<i>Patients with infarcts</i>	
		<i>Number</i>	<i>Per cent</i>
All	5,000	588	11.76
Psychiatric	841	102	12.1
Nonpsychiatric	4,159	486	11.7
White	4,212	541	12.8
Male	3,045	399	13.0
Female	1,167	142	12.2
Negro	788	47	6.0
Male	507	33	6.5
Female	281	14	5.0
All males	3,552	432	12.2
All females	1,448	156	10.8

During 1950 (the median year of the period covered in this study), 67 per cent of the 7,202 patients admitted to the general hospital were men, the average age of the patients was 51.14 years, and the average age of those who came to autopsy was 64.3 years.

The following data concerning sex refer to the 175 patients with old unsuspected infarcts. Old healed unsuspected infarcts were found in 127 men (36 per cent of all autopsies of men), and in 48 women (33 per cent of all autopsies of women). The

average age of the 175 patients was 71.0 years; of the men, 70.6 years (white men, 70.7; colored men, 70.2) and of the women 72.2 years (white women, 72.4, colored women, 69.0). One patient was under 50

years of heart failure were recorded in 42 patients (24 per cent). Thirty of these patients (71.4 per cent) had cardiac hypertrophy at autopsy (Table 30-5), and in 13 patients both hypertension and cardiac hypertrophy were found. In 34 the blood pressure was recorded, 16 of these patients had hypertensive levels, but 24 had cardiac hypertrophy at autopsy.

TABLE 30-3 AVERAGE AGE OF 588 PATIENTS WITH MYOCARDIAL INFARCTS ACCORDING TO TYPE OF INFARCT (FRESH, OLD SUSPECTED, OR OLD UNSUSPECTED), PSYCHIATRIC STATUS, RACE, AND SEX

Type of infarct and group of patients	Patients with infarcts		Average age
	Number	Per cent	
All infarcts	588	100	68.2
Psychiatric patients	102	17.3	70.0
White patients	541	92.0	68.7
Male	399		68.3
Female	142		69.5
Negro patients	47	8.0	61.8
Male	33		64.9
Female	14		54.4
Fresh infarcts	136	23.1	66.9
Psychiatric patients	25	4.3	65.5
White patients	126	21.4	67.6
Male	97		67.0
Female	29		69.7
Negro patients	10	1.7	58.1
Male	7		62.3
Female	3		48.3
Old suspected infarcts	277	47.1	67.0
Psychiatric patients	43	7.3	71.6
White patients	260	44.2	67.8
Male	192		67.6
Female	68		68.6
Negro patients	17	2.9	54.2
Male	9		56.9
Female	8		51.1
Old unsuspected in-			
farcts	175	29.8	71.0
Psychiatric patients	34	5.8	73.4
White patients	155	26.4	71.1
Male	110		70.7
Female	45		72.4
Negro patients	20	3.4	70.0
Male	17		70.2
Female	3		69.0

TABLE 30-4 DISTRIBUTION OF AGE GROUPS IN 175 PATIENTS WITH OLD UNSUSPECTED INFARCTS

Age group	Number of patients	Per cent
Under 50	1	0.4
50-59	23	13.1
60-69	57	32.6
70-79	53	30.3
80 and over	41	23.4
All groups	175	100.0

CEREBRAL THROMBOSIS Physical findings of hemiplegia were noted in 45 cases, and in 7 additional cases cerebral thrombosis or infarction was found at autopsy. In 26 patients the principal lesion was cerebral thrombosis.

TABLE 30-5 FREQUENCY OF HYPERTENSION AND CARDIAC HYPERTROPHY IN ASSOCIATION WITH CONGESTIVE FAILURE AMONG 175 PATIENTS WITH OLD UNSUSPECTED INFARCTS

History or findings of congestive failure	Hypertension	Cardiac hypertrophy
Present (in 24%)	47.1%	71.4%
Absent (in 75%)	31.6	39.8

DIABETES MELLITUS AND GANGRENE. Twelve patients had a history of diabetes, and of these 4 had gangrene of the feet. In addition, arteriosclerotic gangrene of the feet was recorded in 8 nondiabetic patients. In 5 patients gangrene was the principal lesion at autopsy.

ELECTROCARDIOGRAPHIC FINDINGS No electrocardiogram was made in 119 patients. Among the remaining 56 patients, the electrocardiogram was essentially normal in 23 and abnormal in 33. Evidence of left bundle

years (age 49); 23 were aged 50-59, 57, 60-69, 53, 70-79; and 41, 80 or over (Table 30-4).

HISTORY A history of precordial pain or angina was given by the patient at the time of last admission in only 6 patients.

HEART FAILURE Symptoms or physical find-

branch block was noted in 7; in these patients one could not rule out old myocardial infarction

PRINCIPAL CAUSES OF DEATH. In a majority of cases, the presenting complaint or predominant physical findings pointed to some major noncardiac condition. The principal causes of death were judged to be pulmonary disease in 47 (embolism or infarction in 5); cardiac disease in 39 (congestive failure in 29, fresh infarction in 5); neoplastic disease in 34, vascular disease (of brain, or gangrene of foot) in 31; and miscellaneous lesions in 24 (perforated peptic ulcer in 6). Of 29 patients with congestive failure, electrocardiograms were taken in 12. 4 of these were normal, and 8 abnormal but not diagnostic of infarction

DISCUSSION

Barnes and Ball¹ of the Mayo Clinic found myocardial infarcts in 4.9 per cent of a series of 1,000 consecutive autopsies performed prior to 1931. McCain and his associates² at Western Reserve University found an incidence of 7.8 per cent infarcts among 3,559 autopsies performed between 1935 and 1950. The average age of their patients at death was 61.5 years (men, 60.9 years, women, 62.9 years, white patients, 62.2 years, Negro patients, 56.1 years). They encountered myocardial infarcts in 8.69 per cent of men autopsied, and in 6.47 per cent of women autopsied, giving a ratio of men to women with infarcts of 1.32 to 1. The corrected ratio of white to Negro patients with infarcts was about 2 to 1.

PSYCHIATRIC STATUS. The incidence of infarcts among our psychiatric patients (12.1 per cent) was approximately the same as that among our nonpsychiatric patients (11.7 per cent), so that the group of psychiatric patients did not unduly affect the percentage of all patients with myocardial infarcts (Table 30-2).

RACE, AGE, AND SEX. The corrected ratio of white patients with all types of infarcts (12.8 per cent) to Negro patients (6.0 per cent) was 2.13 to 1. The lesser frequency

of infarcts among Negroes compared to white persons and the lower average age of Negroes, particularly of Negro women, are in agreement with the findings of McCain and his associates.

Only 13.7 per cent of 175 patients with unsuspected old infarcts were less than 60 years old; 32.6 per cent were aged 60 to 69, and 53.7 per cent were aged 70 or over.

FREQUENCY OF OLD, HEALED, UNSUSPECTED INFARCTS. It is astonishing that the incidence of old healed "silent" infarcts should be as high as 3.5 per cent of all autopsies. This represents 29.8 per cent (approximately 5 per cent) of all infarcted hearts encountered at autopsy. This finding indicates that myocardial infarct is often well tolerated by the patient. Since the average age of the 175 patients with old unsuspected infarct was 71 years, it is evident that these patients may live out their expected life span.

Weinberg³ of the Medical Examiner's Office of New York City found old myocardial infarcts in 32 per cent of cases of sudden and unexpected death from all types of coronary artery disease. Spain⁴ encountered in the material of the medical examiner's office of Westchester County, New York (a well-to-do residential community close to New York City), old healed myo-

part, were unaware of having had coronary artery disease.

HEART FAILURE. Among patients with a history of findings of heart failure, cardiac hypertrophy was more frequent (71.4 per cent) than among those without a history or clinical evidence of failure (39.8 per cent).

It must be recognized that, following the onset of heart failure, many patients with previous hypertension have reduced or normal levels of blood pressure; in most of these cases one may expect to find a significant increase in the weight of the heart. Likewise, it may be expected that patients with chronic heart failure, regardless of their previous level of blood pressure, will

develop cardiac hypertrophy as a compensatory measure

Patients 50 years of age or over who enter the hospital with chief complaints referable to some organ or system other than the cardiovascular system may have had angina, precordial distress, or heart failure and may forget, neglect, or be unable to give this information at the time of their last admission. The examining physician, centering his attention on the presenting complaints or findings, may well overlook the possibility that the patient has a healed myocardial infarct

CONDITIONS FREQUENTLY ASSOCIATED WITH OLD INFARCTION In patients 50 years or older who enter the hospital with symptoms or signs that do not point directly to disease of the heart, one should nevertheless look for evidence of an old myocardial infarct if any one or more of the following conditions exist: hypertension, history or findings of cerebral thrombosis or hemorrhage, senility, urinary or fecal incontinence, diabetes mellitus, arteriosclerotic gangrene of feet, vascular lesions of eyegrounds, or hypothyroidism.

ELECTROCARDIOGRAPHY Perhaps a careful electrocardiographic examination with multiple unipolar chest leads would have detected the myocardial infarct in most of the 119 patients who had no tracing, as well as in most of those who had a negative tracing, and even in most of those with an abnormal tracing that was not diagnostic of myocardial infarction. In the last group of patients, only one or a limited number of leads was taken

SUMMARY

In order to determine the incidence of old, healed, unsuspected myocardial infarcts among patients dying in a county hospital,

we reviewed the protocols of 3,000 consecutive autopsies from 1945 to 1955, inclusive, and the clinical records of all patients who were found to have infarcts at autopsy. The total number of hearts with infarcts, whether old, recent, or fresh, was 588 (11.76 per cent). The ratio of men to women with infarcts, among those who came to autopsy, was 1.13 to 1; of white to Negro patients, 2.13 to 1.

Unsuspected healed (old "silent") infarcts were found in 175 patients (35 per cent) of all patients who came to autopsy, and constituted 30 per cent of all myocardial infarcts encountered at autopsy.

The majority of the 175 patients with unsuspected healed infarcts (53.7 per cent) were 70 years or older and only 1 was less than 50 years old. Those who had congestive heart failure had associated hypertension, and particularly cardiac hypertrophy, more often than patients who had no evidence of congestive failure.

Electrocardiograms were taken in only 56 of the 175 patients with old unsuspected infarcts. In 33 the electrocardiographic tracing was abnormal but not diagnostic of infarction. Multiple unipolar chest leads probably would have revealed evidence of infarction in many of these 175 patients.

REFERENCES

1. BARNES, A. R., and BALL, R. G. The incidence and situation of myocardial infarction in one thousand consecutive post-mortem examinations, *Am J M Sc* 183:215, 1932.
2. McCAIN, F. H., KLINE, E. M., and GILSON, J. S. A clinical study of 281 autopsy reports on patients with myocardial infarction, *Am Heart J* 39:263, 1950.
3. WEINBERG, S. B. *Personal communication to the authors*.
4. SPAIN, D. M. *Personal communication to the authors*.

CHAPTER 31 ANALYSIS OF THE INCREASE IN MYOCARDIAL INFARCTIONS IN MALMÖ, SWEDEN, 1935-1954

Gunnar Björck

The studies performed by Keys and his collaborators in various parts of the world have shown the importance of environmental factors in the development of atherosclerotic heart disease.¹ The composition of the food apparently does play a great role here. Studies by J. H. Morris in London have pointed to a certain influence, also, of occupation,² and the British mortality statistics demonstrate a new feature in recent years in that their social Class I has shown an increase in mortality rate over social Class II. Malmros, Strom, and I have, in various connections, drawn attention to the marked influence of wartime conditions in Scandinavia upon mortality rates for various diseases, including those of the heart and circulation.³ There was a decrease in mortality in all age groups, particularly in the years 1942-1943, when food rationing was most severe, and the mortality rates rose again at the return of living conditions to previous levels or higher.

During the last five years, my collaborators

and I have tried to collect information on the epidemiology and natural history of certain cardiovascular diseases in Malmö, the town where I live. It is the third largest city in Sweden, with 200,000 inhabitants, and located opposite Copenhagen in the south of the country. Most people are employed in industry or business. In this city, there is only one large, general hospital, which at the same time is a teaching hospital for the University of Lund, located nearby. There are no private hospitals and, consequently, the hospital population is truly representative of the population of the city, whose composition is well known statistically. The autopsy rate for the department of medicine over the last 20 years has ranged between 90 and 97 per cent. I believe there are few places in the world where similar conditions exist for epidemiological work on a clinical level. Therefore, we have been anxious to utilize this material in order to study, among others, the following questions: Can the apparent increase in incidence of myocardial infarction

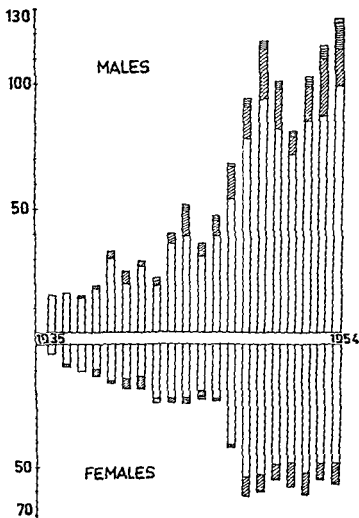
be traced back to some particular features in the population, such as the relative increase in the number of older people, or a change in the distribution of the population with regard to occupation? Furthermore, is the increase in incidence of myocardial infarction evenly distributed between men and women or over the period of observation? Is the number of infarcts in the population unchanged, and only the fraction who are being hospitalized on the increase? Is there a change in the age at the first infarction? Is there a change in severity of the infarcts? Is there a change in mortality rate over the years? Has anticoagulant treatment decreased the mortality rate? Are second and third infarcts more frequent nowadays? How

good was the clinical diagnosis of myocardial infarction in the light of autopsy examination?

MATERIAL

The material I have been reviewing, together with Drs Gunnar Blomquist and Jan Sievers, comprises 1,612 patients with a total of 1,825 infarcts, who were treated in our hospital between 1935 and 1954. This is a large bulk of material, and I will not discuss the clinical findings nor the follow-up studies on the 94 per cent of the patients whose course we have been able to follow. I will restrict myself to brief answers to some epidemiological questions.

FIG 31-1 Incidence of hospitalization for myocardial infarction in Malmö in years 1935 to 1954, in both men and women. Note the increase during the 20-year period. Open areas represent first infarctions, diagonal cross-hatching indicates second infarcts, horizontal cross-hatching shows third infarcts, vertical cross-hatching designates fourth infarcts, and solid areas represent fifth infarcts.



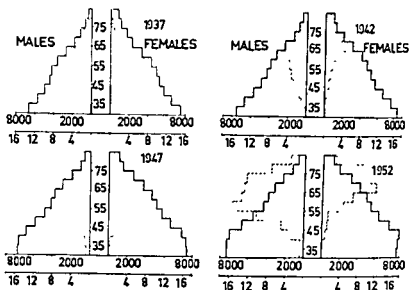


FIG 31-2 Increase in incidence of myocardial infarction in Malmö projected upon the population distribution according to age. Solid outline and upper scales represent the distribution of males and females by 5-year groups. The dotted lines and lower scales represent the patients hospitalized with myocardial infarctions in the corresponding age groups.

INCREASED INCIDENCE OF INFARCTION

Over these 20 years there has been a considerable increase in the number of patients hospitalized for myocardial infarction (Figs

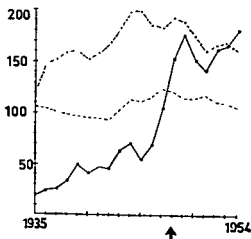


FIG 31-3 Availability of hospital beds in relation to incidence of myocardial infarction. Solid line represents total number of patients with myocardial infarction. Dashed line indicates number of beds available in the Department of Medicine per 100,000 inhabitants. Dot-dashed line shows number of admissions, annually, to the Department of Medicine, per 10,000 inhabitants. Solid arrow indicates beginning of anticoagulant therapy.

31-1 and 31-2). It is obvious that the increase concerns both men and women, that it is far in excess of what can be expected from the increase of older age groups in the population, and that the increase is not quite linear. There are two periods of retardation, one in connection with the war years, and one at the end of the period. The years 1947 and 1948 represent a steep increase from one level to another one.

A primary problem here is the availability of hospital beds in the Department of Medicine. The number of occupied beds per 1,000 inhabitants has changed as seen in Figure 31-3. We do not feel that the increase in patients with myocardial infarction is related to the number of hospital beds available, because there has always been room in the hospital for patients of this type. The sigmoid shape of the curve for the incidence of myocardial infarction may be due rather to a release of postponed infarcts in the early forties, or to a change in admittance policy on the part of the medical practitioners.

As a matter of fact, our study faces its greatest difficulties in establishing the importance of this factor of selection for hospitalization, and so, probably, do all studies of this kind. We have attempted to approach

the question from three points of view, viz : (1) the composition of the clinical material, (2) the occurrence of acute cardiac infarcts and localized myocardial fibrosis (*infarct scar*) in the *total* autopsy material in the city, (3) and direct questioning of the practitioners. With regard to the two first, *objective* investigations, we have thought that a change in the policy of admittance would be reflected in the composition of the hospital material, probably by a greater frequency of mild cases or of very severe cases, with corresponding effects on mortality; a *real* increase in myocardial infarction would, furthermore, be reflected in a higher incidence of myocardial fibrosis in the *total* autopsy material, as well as in the number of patients dying from acute myocardial infarctions.

AGE AND SEX

The composition of the material with regard to age, men and women, is seen from Figures 31-4 and 31-5. Both men and women show an increase in mean age, more so for women than for men. This increase obviously exceeds the simultaneous increase in mean age of the population. The number of infarcts has not increased by an accumulation of many young infarcts, but, on the contrary, of old infarcts. Neither can the increase be explained on a *sex ratio* basis (Fig. 31-6). The mean sex ratio of males to females in

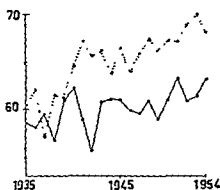


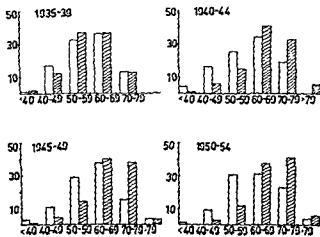
FIG. 31-4 Mean age of patients admitted to the hospital with initial infarctions. Males indicated by solid line, females shown by dotted line.

our material is 1.66:1. This figure is in good agreement with other Swedish figures, but lower than American ones.

OCCUPATION

In this connection, some remarks should be given on *occupation*. Data on occupation are difficult to handle. First of all, many people pass through various occupations and social classes during their lifetime, and many are struck by their infarcts after retirement (30 per cent of the men in our material). Furthermore, the mean age in the various occupational groups is different. Lastly, the numbers must be projected against the distribution of the occupational groups in the

FIG. 31-5 Distribution of patients with myocardial infarction according to age and sex, in 5-year periods from 1935 through 1954. Males shown in open columns, females indicated by cross-hatched columns.



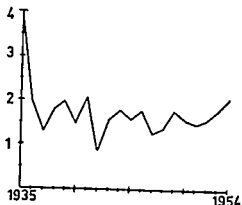


FIG 31-6 Ratio of males to females in 1612 patients with myocardial infarction hospitalized between 1935 and 1954

society In our city, however, there is little reason to assume differences in hospitalization rates between the occupational groups or social classes

Figures 31-7 and 31-8 show the incidence of myocardial infarction expressed as number of cases per thousand male employers, clerks, and workers in the population of Malmö. Using χ^2 analysis, no significant differences exist in the first 5-year period. The incidence among employers is significantly higher than for workers in the second period, and this is true, also, in comparison to clerks during the third and fourth period. The higher incidence among clerks over 65 years in the second 5-year period is striking in the diagram, but the material is small. In the fourth period, the difference is significant in *all* age-groups. There are no significant differences between clerks and workers. Figures that are considered somewhat less certain indicate that corresponding trends also occur among wives of employers,

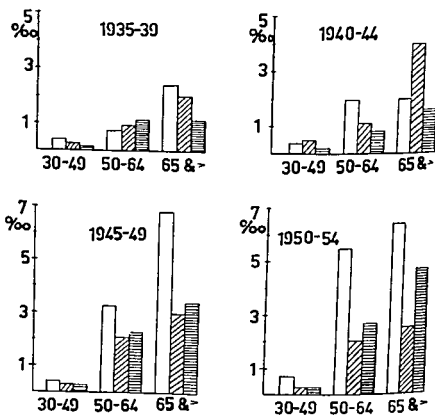
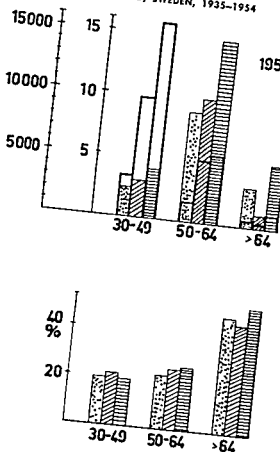


FIG. 31-7. Incidence of primary myocardial infarction in men of various occupational classifications in Malmö by 5-year periods from 1935 through 1954. The chart illustrates the number of myocardial infarctions per 1,000 inhabitants in three age groups within the occupational groups. Open areas indicate employers, diagonal cross-hatching represents clerical workers, and horizontal cross-hatching shows workers.

FIG 31-8 *Top* Frequency of myocardial infarction in various occupational groups, projected upon the population of employers, clerks, and workers, respectively, in various age groups in Malmö (left scale) Dotted areas represent employers, diagonal cross-hatchings indicate clerical workers, and horizontal cross-hatching indicates workers. The average number of myocardial infarctions per year from 1950 to 1954 in the various age and occupational groups is shown. *Bottom* Acute mortality percentage, by age and occupational groups, due to initial myocardial infarction from 1935 to 1954 in entire material studied. Occupational groups designated as for top graph.



clerks, and workers. These findings are in accord with those of Ryle and Russell,⁴ but not with some American reports.

TYPE OF INFARCTION

The question of the clinical type of the infarcts has been analyzed, and a classification made as in Figure 31-9. Group I contains the very severe cases, generally admitted in a state of shock, Group II the moderately severe cases, and Group III the mild cases. Group IV contains patients with a typical clinical picture but without definite electrocardiographic evidence of infarction, and Group V cases are those wherein the diagnosis was first made at autopsy. The percentages here indicate that there has been no significant change in the composition of the patient material, hospitalized because of infarction, with regard to severity, during these 20 years. The mortality also has shown

a certain increase in the period 1940-44, which is difficult to explain, and there has been a significant decrease in mortality for males in the last 5-year period. The average mortality in primary infarcts was 32 per cent for men and 35 per cent for women (Fig 31-10).

From the evidence presented it emerges, that there has been an increase in the number of hospitalized infarcts over this 20-year period, which is in excess of what would be expected from data on age-group composition of the population. The curve is not linear but shows a sigmoid shape with a particularly steep increase in the years 1947-48. In the material of hospitalized patients, there is a selective increase in the proportion of older patients but no significant trends with regard to sex ratio, degree of severity of the clinical condition, or mortality during the period. There is, furthermore, a tendency to increasing morbidity but not of mortality in

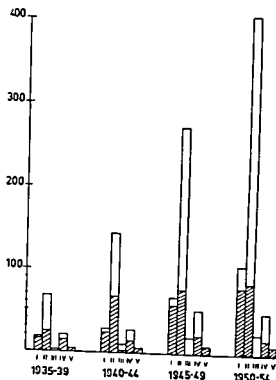


FIG 31-9 Distribution of patients with primary infarction according to clinical type by 5-year periods. Group I, severe with shock, Group II, moderately severe, Group III, mild, Group IV, typical clinical picture without electrocardiographic evidence, Group V, diagnosis at autopsy. Cross-hatched area indicates number of deaths.

myocardial infarction in the group of employers in relation to employees during the period

ACCURACY OF DIAGNOSIS

Statistics on the causes of death in the city over the 20-year period are hardly useful for our purposes, as they may reflect chiefly changes in terminology, particularly following the adoption of the new World Health Organization nomenclature in 1951. We have, however, been looking into the autopsy material of the city. The autopsy rate at the hospital is rather high over the 20-year period, but this is not true of autopsies from persons dying outside the hospital. The rate of such autopsies is, however, on the increase during the period 1944-54, the only one from which full data are obtainable. The percentage of autopsies to all deaths was 28.9 per

cent in 1944 and 41.5 per cent in 1954. The hospital autopsies account for 84 per cent of all autopsies in the period 1944-54. We have looked into the accuracy of the clinical diagnosis of myocardial infarction in the hospital, as judged from the autopsy report, as seen in Table 31-1. There has been an improvement

TABLE 31-1 ACCURACY OF CLINICAL DIAGNOSIS

Years	Number of patients with acute myocardial infarct at autopsy	Under-diagnosis		Over-diagnosis	
		Number	Per cent	Number	Per cent
1935-39	58	9	15.5	9	15.5
1945-49	204	21	10.3	19	9.3
1950-54	256	26	10.2	9	3.5
Total	518	56	10.8	37	7.1

in diagnostic accuracy, which is particularly evident under the classification of *overdiagnosis*. This observation signifies that the number of nonfatal clinical infarcts at the beginning of this 20-year period may have been

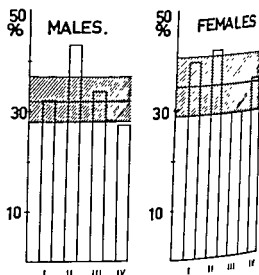


FIG 31-10 Mortality rate due to myocardial infarction in males and females. Mortality rates per 5-year periods are shown in Column I, 1935 to 1939, Column II, 1940 to 1944, Column III, 1945 to 1949, and Column IV, 1950 to 1954. Cross-hatched zone shows mortality rate for entire material from 1935 to 1954, with 3X standard error limit indicated.

even smaller than reported, and that the observed increase probably is not due to improved clinical diagnosis in the hospital

The statistics for the total autopsy material for the years 1944 to 1954 are summarized in Figure 31-11. This figure shows that the rise in autopsy diagnosis of *acute myocardial infarction* in the city closely follows the increased autopsy rate of the period 1944-54, and that when these figures are expressed as percentage of myocardial infarction of the total of all autopsies, *no increase* over this period is seen. However, a closer study reveals that the increase in total autopsy rate derives chiefly from hospital departments other than the Department of Medicine. Therefore, the absolute figures are probably more representative than the relative ones, and support the impression from the hospital material of an increase in incidence of acute myocardial infarction. Unfortunately, we have not been able to recover the corresponding material from the first half of the 20-year period.

LOCALIZED MYOCARDIAL FIBROSIS

The incidence of *localized myocardial fibrosis* (infarct scars) in the total autopsy

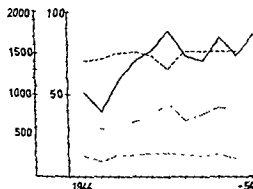


FIG 31-11 Total autopsy material for the city of Malmö from 1944 to 1954. Dashes show total number of deaths (left scale). Dots indicate frequency of autopsy examination, percentage of all deaths. Solid line indicates total number of cases of acute myocardial infarction disclosed at autopsy. Dots-dashes represent frequency of acute myocardial infarction, percentage in all autopsied cases.

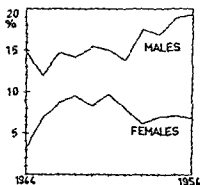


FIG 31-12 Incidence of *localized myocardial fibrosis* according to sex in the years 1944 through 1954. The diagram indicates the percentage disclosed in all cases autopsied at the hospital. Data in Figs 31-11 and 31-12 refer only to patients over 30 years of age.

material over the years 1944-54 shows a rise for men, both absolute and relative, but no clear tendency for women (Fig 31-12). Although the differences in the figures for men are significant for the material from the Department of Medicine, they are not for the total material. Probably tendencies in this regard will manifest themselves more slowly and over a longer period of time than is the case with acute infarcts. Thus, we are faced with the picture of a marked increase in clinical myocardial infarction, which is only partly corroborated in the autopsy material at our disposal.

Therefore, we have turned to the medical practitioners in the city to find out whether, in their opinion, improvement in their ability to make the diagnosis of myocardial infarction, and/or a different attitude towards hospitalization of such cases, might be responsible for an increase in hospitalization rate of patients with myocardial infarction rather than in the total incidence of myocardial infarction. The general opinion was that, among the younger generation of practitioners, the diagnostic accuracy was already satisfactory at the beginning of the period, whereas the older practitioners probably were slow to differentiate between angina pectoris and myocardial infarction up to the early 1940's. As for the attitude toward hospitalization during the whole period, most of the

practitioners have admitted all patients with myocardial infarction to the hospital. A few, however, state that they treated more persons at home twenty years ago than now, because people were more easily cared for by the family in previous years than now. These practitioners feel that, today, everyone who develops an infarction is sent to the hospital. Another few practitioners stated that they changed their policy in the late 1940's when anticoagulant treatment was introduced at the hospital (1948), and they felt obliged to avail all their patients of this treatment.

The information obtained can be summarized thus: (1) improved diagnosis may account for a part of the rise in the incidence of myocardial infarction up to the early 1940's but this does not explain, specifically, the steep increase during the years 1946-47. (2) In the last five years of the 20-year period, practically all patients with myocardial infarction were diagnosed and brought to the hospital. This may have some relation to the fact that the present figures suggest a *steady state*.

The analysis of our material is not yet complete. Therefore, I will restrict myself to the presentation of the data, and will not now go more deeply into interpretations. However, I hope that the data per se have been of some interest to this conference.

BIBLIOGRAPHY

1. KEYS, A., ANDERSON, J. T., ARESU, ..., BJORCK, G., BROCK, J. F., BRONTE-STEWART, B., FIDANZA, F., KEYS, M. H., MALMROS, H., POPPI, A., POSTELL, T., SWAHN, B., and DEL VECCHIO, A.: Physical activity and the diet in populations differing in serum cholesterol, *J. Clin. Invest.* 35 1173, 1956.
2. MORRIS, J. N., and RAFFLE, P. A. B.: Coronary heart disease in transport workers: a progress report, *Brit. J. Indust. Med.* 11 260, 1954.
- 3a. MALMROS, H.: Relation of nutrition to health: statistical study of effect of war time on arteriosclerosis, cardiosclerosis, tuberculosis, and diabetes, *Acta med. scandinav. supp.* 246, p. 137, 1950.
- 3b. STRÖM, A.: *The Influence of Wartime on Health Conditions in Norway*. Institute of Social Medicine, University of Oslo, 1954.
- 3c. BJÖRCK, G.: Social significance of cardiovascular diseases in Sweden, *Acta med. scandinav. supp.* 259, p. 163, 1951.
- 3d. BJÖRCK, G.: "Wartime Lessons on Arteriosclerotic Heart Disease from Northern Europe," in *World Trends in Cardiology*. A. KEYS, and P. D. WHITE (Eds.), 1 *Cardiovascular Epidemiology*, New York, Hoeber-Harper, 1956, pp. 8-21.
4. RYLE, J. A., and RUSSELL, W. T.: Natural history of coronary disease: a clinical and epidemiological study, *Brit. Heart J.* 11 370, 1949.

CHAPTER 32 RELATION OF SEX, AGE, AND PHYSICAL ACTIVITY TO SUDDEN DEATH FROM CORONARY ARTERIAL OCCLUSION

David M. Span and Victoria A. Bradess

The question of physical activity as related to coronary artery disease has resolved itself into several separate problems. Firstly, the relationship of activity or lack of it to the pathogenesis of the atherosclerotic process; secondly, the relationship of physical exertion to the precipitation of the manifestations of acute coronary occlusion; and, lastly, the effect of activity or lack of it on the myocardium—recently or remotely damaged from pre-existing coronary atherosclerosis with or without occlusion.

This report is concerned with the first two aspects of the problem, based on a study of over 1,000 consecutive cases of sudden death due to coronary artery disease (The Negro cases are not included in this portion of the study.) These constituted the entire group of cases reported to the Medical Examiner's office in Westchester County over a period of 6 years. Most were those cases of sudden

death from coronary artery disease that either did not have any previous medical care, had no evident symptoms, had died in a public place, or were without attendance from a physician. Westchester County is a community of 675,000 people, and contains representative small cities, towns, villages, industrial, residential, semirural, and suburban areas. With few exceptions, these cases were residents of Westchester County. The sex and age distribution of these cases may be noted in Table 32-1. Of the 867 male cases, 652 had an adequate and verified occupational history and the age was known to be accurate. The diagnosis of death due to coronary artery disease was verified at autopsy in about half of these cases. In the remainder, the previous clinical and medical evidence, as well as a characteristic picture of attack leading to death, were regarded as sufficiently conclusive. If there

TABLE 32-1. DISTRIBUTION ACCORDING TO AGE AND SEX
(White Cases)

	Under 35	35-40	41-45	46-50	51-55	56-60	61-65	66-70	Over 70
Male	13	38	57	81	120	149	133	133	143
Female	3	4	2	10	12	23	42	37	83

was the slightest doubt as to the diagnosis, an autopsy was performed. The occupations were graded into three groups as regards activity: sedentary, moderate activity, and considerable activity. Illustrations of those classified in the sedentary group were bus drivers, bank executives, accountants, and stenographers. Examples of moderate activity were traffic officer, electrician, and train conductor. Some examples of those with considerable activity would be steam fitter, ditch digger, heavy construction worker, and sanitation truck loader. These were then classified into two age groupings, those under 55 years and those 55 years of age and over. In Table 32-2 it may be noted of the 251 cases

them were listed as housewives, and it was impossible to determine the variations in activity. The limitations of this type of analysis are obvious, since no information was available concerning extraoccupational time relative to physical activity.

These findings are in accord with most recent observations concerning relationship of physical activity as evidenced by occupation with the development of coronary atherosclerosis. The proper interpretation of these findings is another story. This association does not necessarily imply that it is the physical activity or lack of it per se that is the factor. There may be different stress situations associated with sedentary versus physically active jobs, dietary habits may differ, and the factor of self-selection cannot be excluded. The correct evaluation and interpretation of these findings await much more intensive and carefully controlled investigation on a broad epidemiological scale.

There is considerable overlap of the factors with coagulability of the blood and lipid metabolism as they relate to coronary artery disease. Thus, one finds conflicting ideas concerning the early pathogenesis of the lesion, especially in views postulated by Duguid² that mural vascular fibrin thrombi are often regarded as the initial nidus of the atherosclerotic lesion as opposed to the more widely held lipoprotein complex concept. Heparin is involved not only in blood coagulation, but is also concerned with serum lipid clearance. Fatty meals, on the one hand, may be related to cholesterol levels and, on the other hand, may cause a postprandial increase in blood coagulability. Unsaturated fatty acids may exert a cholesterol-lowering effect, as claimed by some, and also may increase fibrinolytic activity. The question of the relationship of the acute thrombotic episode to the under-

TABLE 32-2. DISTRIBUTION OF WHITE MALES WITH ACUTE CORONARY OCCLUSION ACCORDING TO OCCUPATIONAL PHYSICAL ACTIVITY AND AGE

Age	Sedentary activity	Moderate activity	Strenuous activity
Under 55	112 (44%)	81 (31%)	35 (24%)
Over 55	139 (56%)	179 (69%)	105 (76%)
Totals	251 (100%)	260 (100%)	141 (100%)

listed in the sedentary activity group, 112 (44 per cent) died from their coronary artery disease under the age of 55 years, whereas those in the moderate activity group, 81 of 260 (31 per cent), and those in strenuous activity only 35 out of 141 (24 per cent) died under the age of 55 years. Thus, it may be noted that, with an increase in physical activity as related to occupation, there was a trend toward a smaller number of cases dying from coronary artery disease, proportional to the total number in each group under the age of 55 years. The female cases were not tabulated in this manner, because most of

lying atherosclerosis is obscured by this overlap and is consequently not completely resolved. Are there factors independent of the atherosclerotic process that lead to thrombosis in the coronary arteries, or is the thrombus merely a complication occurring unpre-

cardial infarcts, evidence of any other form of heart disease, the degree of atherosclerosis, and presence of thrombi. These findings were related to age, sex, and physical activity prior to the attack that led to sudden death. In Table 32-3 it may be noted that one out of

TABLE 32-3 SEX INCIDENCE AND PHYSICAL ACTIVITY PRIOR TO DEATH IN ATHEROSCLEROTIC AND THROMBOTIC CORONARY OCCLUSION (410 AUTOPSED CASES)

Type of occlusion	Number of cases	Male	Female	Number of cases with
				physical activity prior to attack
Atherosclerotic	310	277	33 (10.6%)	50 (13%)
Thrombotic	100	90	10 (10%)	17 (17%)
Total	410	367	43	67

dictably in the course of advanced coronary atherosclerosis? What is the relationship of physical exertion to the precipitation of sudden fatal coronary occlusion, whether this be atherosclerotic alone or in combination with an acute thrombosis? In order to evaluate this latter problem, 410 consecutive autopsied cases in which death had occurred suddenly from coronary artery disease, and in whom there was available a reliable history of the

every four cases had a thrombus. This same ratio persisted when the cases were divided according to sex. Of the 410 cases, 67 had a definite history of physical activity that could be classified as moderate to strenuous in the period immediately preceding the onset of the attack. When this type of physical activity was related to the atherosclerotic and the thrombotic grouping, no significant difference in incidence was noted.

TABLE 32-4 AGE DISTRIBUTION OF ATHEROSCLEROTIC AND THROMBOTIC CORONARY OCCLUSION Males, 364 Autopsied Cases

Type of case	Under 39	40-49	50-59	60-69	Over 69
Atherosclerotic occlusion	35	78	84	58	20
Thrombotic occlusion	5 (12.5%)	26 (25%)	28 (25%)	24 (34%)	6 (23%)
Total	40	101	112	82	26

type of physical activity just prior to the acute episode, were analyzed. These were part of the original 1,000 Medical Examiner's cases and were divided into two groups—those with advanced atherosclerosis alone and those with superimposed recent thrombi. Coronary arteries were cross-sectioned at several millimeter intervals in all the main branches throughout their entire length. In many of these cases, as part of another study, the Schlesinger injection technique² had been used. The heart weights were recorded as well as the presence of old and recent myo-

In Table 32-4, the age distribution of the atherosclerotic and thrombotic coronary occlusions in the male group is recorded. In this table it can be seen that under the age of 39 years, thrombotic occlusion was present in 12.5 per cent of the group and increased in succeeding decades so that in the age group of 60 to 69 years it was 34 per cent. There were too few female cases for significant grouping by decades. However, these were divided into two groups with the age 50 years as the dividing point. This age was taken as an approximation of the menopausal

period. Although the number of cases (13) was too small to allow any conclusions, no significant differences in the sclerotic-thrombotic ratio was observed in the two age groups (Table 32-5)

TABLE 32-5 ATHEROSCLEROTIC AND THROMBOTIC CORONARY OCCLUSION
AGE DISTRIBUTION
Females

Type of case	Under 50	50 and over
Atherosclerotic occlusion	7	24
Thrombotic occlusion	3	9
Total	10	33

In Table 32-6 the incidence of recent and old infarcts in both groups as well as their relationship to physical activity prior to the sudden fatal attack are noted. Here, again, no significant differences between the atherosclerotic and thrombotic groups are observed. Furthermore, moderate to strenuous physical

TABLE 32-6 RELATIONSHIP OF TYPE OF OCCLUSION, INFARCTS, AND STRENUOUS PHYSICAL ACTIVITY TO ONSET OF SUDDEN FATAL ATTACK
(367 Autopsied Males)

Type of occlusion	Number of cases with infarcts	Number of cases strenuous activity	Number of cases activity and infarcts
Atherosclerotic	277	68 (25%)	41 (6)*
Thrombotic	90	31 (34%)	15 (2)*
			8 (53%)

* 6 and 2 cases, respectively, engaged in strenuous activity in contrast to usual occupational activity

activity as a possible precipitating factor was no more frequent in the cases with infarcts than in the entire group studied, with perhaps one exception. The possible exception is that group of cases in which thrombosis and infarction were both present. In this situation over 50 per cent of the cases had a history of previous physical exertion. The number of cases involved in this group is too small to make any conclusions whatsoever. It is also possible that, because of the nature of the autopsy findings, the recorded activity oc-

curred after the occlusive event rather than prior to it. It is also of interest to note that only a very few individuals were engaged in strenuous activity not related to their usual occupational activity.

These findings would favor the concept that coronary thrombosis is a complication occurring during the course of coronary atherosclerosis rather than an independent process. If other independent factors were involved, one might not expect such a similar sex distribution and physical activity incidence in the two groups, namely the atherosclerotic and the thrombotic. The age ratio also favors this concept, since one would obviously expect an increasing proportion of thrombotic cases, the longer the sclerotic process existed. The findings in the coronary arteries relative to the atherosclerotic-thrombus ratio in this group of cases differs entirely from that seen in coronary artery disease in the usual hospital autopsy series where thrombosis superimposed on atherosclerosis outnumbers cases of death from coronary artery disease without thrombosis. The only obvious difference in the Medical Examiner's cases, as compared with the hospital cases, is the time element between the onset of the attack and death. Does this perhaps signify that a thrombus is secondary to a pathophysiological or occlusive event rather than a precipitating factor? Although this study was not designed with the technique and care necessary for the evaluation of intimal or mural hemorrhages as a factor related to thrombus formation, gross intimal or mural hemorrhages were seen only rarely in the atherosclerotic group.

SUMMARY AND CONCLUSIONS

1 Sedentary type of occupational activity is associated with an earlier onset of death from coronary artery disease

2. Strenuous physical activity or exertion

not appear a factor of any significant consequence. This appears to be so regardless of

whether the occlusion is atherosclerotic or thrombotic.

3 The findings in this report support the concept that acute coronary thrombosis is a complication of, and intimately related to, the atherosclerotic process rather than being caused by independent factors

REFERENCES

1. DUGAN, J. B.: The etiology of atherosclerosis, *Practitioner* 175:244, 1955.
2. SCHLESINGER, M. J. An injection plus dissection study of coronary artery occlusions and anastomoses, *Am. Heart J.* 15 528, 1938

CHAPTER 33 CIRCUMSTANCES RELATED TO SUDDEN, UNEXPECTED DEATH IN CORONARY HEART DISEASE

Sidney B. Weinberg and Milton Helpern

Heart disease, particularly coronary artery disease, is the most frequent cause of sudden and unexpected natural death. The unexpected nature of these deaths in apparently healthy individuals, arising from a variety of circumstances, has evoked considerable interest in what the individual was doing prior to and at the time of his fatal heart attack. The possibility of a causal relationship, in the sense of a precipitating factor, between the deceased's preceding activity and his death has become an important consideration in the evaluation and adjudication of cardiac fatalities by Workmen's Compensation Boards. The importance of this problematical relationship is evident when one considers that this disease, without warning, affects many of the most active and productive individuals in the prime of life in all strata of our society.

PRESENT STUDY

The present study of correlation between circumstances and sudden, unexpected death is based upon 428 consecutive autopsied cases of coronary artery disease in the Office of the Chief Medical Examiner, Borough of Manhattan, for the 5-year period from January 1, 1951, through December 31, 1955. The causes of death in these cases include those from coronary arteriosclerosis alone and coronary arteriosclerosis complicated by acute and/or old thrombotic occlusion, with or without myocardial infarction and fibrosis.

The Medical Examiner's Law in New York City provides for the investigation and certification by the Medical Examiner's Office of the cause of all sudden, unusual, and suspicious deaths. In deciding which of these cases should be autopsied, the circumstances are an important consideration. Au-

autopsies are more apt to be performed in apparently healthy younger individuals in order to detect or rule out physical violence or chemical poisoning. In the older group, objection from the next-of-kin is more likely. This unequal selection of cases for autopsy obviously affects the percentage distribution of the deaths in the various age groups. Nevertheless, a considerable number of older individuals dying from coronary artery disease are autopsied. It is also becoming more frequent for relatives of persons dying from natural disease to request post-mortem examinations in order to learn the cause of death. In recent years it has become increasingly important to provide accurate anatomical information concerning the nature and extent of heart disease. The work of the referee or a court and jury is rendered easier if the precise character of the lesion in a fatal case of heart disease is available for information, especially in connection with continuing claims arising out of past episodes of coronary artery disease in which there was a question of relationship to an exertional incident.

RESULTS

Tables 33-1 and 33-2 summarize the incidence of death from coronary artery disease according to race, sex, age, and the percentage distribution of the population in Manhattan.¹ Of the 428 cases, the youngest per-

TABLE 33-1 DISTRIBUTION OF SUDDEN AND UNEXPECTED DEATH FROM CORONARY HEART DISEASES ACCORDING TO RACE AND SEX

	Number of cases	Population percentage (Manhattan)
White	374 (87.4%)	79
Colored	48 (11.2%)	19.6
Others	6 (1.4%)	1.4
	Number of cases	Population percentage
Males	392 (91.6%)	48
Females	36 (8.4%)	52
White	29 (6.8%)	
Colored	7 (1.6%)	

TABLE 33-2 DISTRIBUTION OF SUDDEN AND UNEXPECTED DEATHS FROM CORONARY HEART DISEASES ACCORDING TO AGE INCIDENCE BY DECADES

	Number of cases	Percentage	Percentage of population (Manhattan)
3rd decade	9	2.1	17.03
4th decade	47	10.98	17.09
5th decade	98	22.9	16.47
6th decade	153	35.7	13.77
7th decade	93	21.7	9.08
8th decade	27	6.3	4.44 (70-84 yrs)
9th decade	1	23	

son was twenty years old and the oldest was eighty years of age. The incidence as to race, sex, and age are comparable to reports in the literature.

The classification of occupation in Table 33-3 is the one used by Master.^{2,3} Distribu-

TABLE 33-3. SUDDEN AND UNEXPECTED DEATHS FROM CORONARY HEART DISEASE CLASSIFICATION OF OCCUPATIONS (428 Cases)

	Number of cases	Percentage of working population in New York City
Workers and laborers	248 (58%)	50
Storeworkers, white collar and businessmen	78 (18%)	39
Professional class	45 (11%)	11
Total	87%*	100

* The remaining 13 per cent (57 cases) consists of housewives, retired workers, and those whose occupations are unknown.

tion of the professional group and that of the workers and laborers in the series is closely related to the percentage population of these various occupations in New York City. The disparity in the percentage of store, office, and business personnel and that of the population distribution may be because many of these people die unexpectedly at their place of employment in the presence of witnesses. The circumstances surrounding the death do not provide enough basis for the arbitrary performance of necropsy by the Medical Ex-

aminer when strenuous objection is raised by the next of kin. In the working and laboring class, the question of causal relation of effort, strain, and trauma to the fatal heart attack is always present. It is in this group that the family is more likely to acquiesce or even request autopsy. In these cases, traumatic injury suggests itself to the family as a reasonably possible cause of death, however minor or remote the injury may be.

TABLE 33-4 TYPES OF PHYSICAL ACTIVITY AT TIME OF FATAL HEART ATTACK (428 Attacks)

	Number of cases	Percentage
1 Sleep	54	12.6
2 Resting		
a Lying down	16	3.7
b Sitting	14	3.3
3 Ordinary mild activity (except walking)	158	36.9
4 Walking	52	12.1
5 Moderate activity	17	4.0
6 Marked activity	9	2.1
7 At work		
a Mild activity	40	9.3
b Moderate activity	39	9.1
c Marked activity	29	6.8

Table 33-4, patterned after Master,² indicates the type of activity at the time of the fatal heart attack. This table reveals that 19.6 per cent of the deaths occurred during sleep or rest. In many instances it is impossible to ascertain the activity of the individual prior to rest or sleep. The largest percentage of deaths, 36.9 per cent, occurred during ordinary mild activity, exclusive of walking. Ordinary mild activity includes talking, gesturing, and standing. Of the total, only 8.9 per cent died during marked activity, either at work or otherwise. Lifting or unloading heavy objects, fire fighting, and running for a bus are examples of marked activity. Moderate activity includes those who were shopping, driving an automobile or taxi in city traffic, and operating a floor buffer. Death occurred in 12.1 per cent while walking in the street. Fatal heart attacks occurred in 25.2 per cent while the individual was en-

gaged at his occupation. It is difficult to obtain an accurate history from witnesses from relatives of the deceased concerning activity at the time of death and frequently the circumstances are unknown in those cases in which the deceased is found dead. Of 147 per cent of the cases were known to have had clinical symptoms prior to death, i.e., sudden chest pain, dyspnea, or complaint of feeling ill.

In discussing activity in relation to fatal heart attack, emotional factors are important, independent of or in association with exertional factors. In many cases, emotional factors appear to dominate. The intangible and immeasurable nature of these factors makes them exceedingly difficult to evaluate and therefore they are often ignored. Yet they cannot exclude their presence and possible effect as exemplified by the following case histories. Of course, the possibility of coincidence cannot be denied.

TABLE 35-5A. POST-MORTEM FINDINGS, SUDDEN AND UNEXPECTED DEATH FROM CORONARY HEART DISEASE (428 Cases)

	Number of cases
I Progressive atherosclerosis (sclerotic occlusion)	296 69
II. Thrombotic occlusion	132 31
Total thrombotic occlusions (147)	
a Recent occlusion (95)	
b Old occlusion (52)	
III Myocardium	31 7
Acute infarction	14 3
Acute and old infarction	13 3
Rupture	137 32
Old infarction	100 23
Focal fibrosis	133 31
No myocardial lesions	

1. A 63-year-old white male, delicately built, owner, was held up and collapsed immediately after the robbery. Autopsy revealed a 500-gm heart. The coronary arteries showed extensive atherosclerosis. An old infarction was noted on the septal and posterior walls.

2. A 58-year-old white male salesman was

being questioned in a stationhouse concerning the theft of a suit when he collapsed. Autopsy revealed a 550-gm heart. The coronary arteries showed severe atherosclerosis. Focal fibrosis was noted in the septal wall.

TABLE 33-5B. POST-MORTEM FINDINGS

	Number of cases	Per- centage
I Progressive atherosclerosis (sclerotic occlusion)	296	69
A Myocardium		
Acute infarction	10	3
Acute and old infarction	6	2
Rupture	5	2
Old infarction	88	30
Focal fibrosis	78	26
No myocardial lesions	109	37
II Thrombotic occlusion	132	31
A Recent occlusions (95)	88	21
Myocardium		
Acute infarction	19	22
Acute and old infarction	8	9
Rupture	8	9
Old infarction	22	25
Focal fibrosis	10	11
No myocardial lesions	21	24
B Old occlusions (52)	44	10
Myocardium		
Acute infarction	2	5
Old infarction	27	61
Focal fibrosis	12	27
No myocardial lesion	3	7

3 A 49-year-old white male, baker, collapsed while at work in a restaurant. His friend, a 53-year-old white male employed as a cook in the same establishment, also collapsed on seeing the man lying on the floor. Both were born in Germany and had been lifelong friends. Autopsy revealed that each had severe coronary atherosclerosis. The myocardium of each heart showed patchy fibrosis. The baker's heart weighed 540 gm and the cook's heart weighed 460 gm.

4 A 65-year-old white female, entering her place of employment, was stopped by two union organizers who attempted to talk to her. As she tried to avoid conversation she collapsed. Her son-in-law stated that she had been frightened by the organizers one week previously. Autopsy revealed a 430-gm heart. The coronary arteries showed severe progres-

sive atherosclerosis. The myocardium showed no evidence of scarring.

Tables 33-5A and 33-5B summarize the post-mortem findings of the 428 cases in the series. In 296 cases (69 per cent) the significant lesion was a gradually progressive coronary atherosclerosis of varying segmental extent and without thrombotic occlusion. In this group, old myocardial infarction and focal fibrosis were seen in 166 of the hearts (56 per cent). No myocardial lesions were noted in 109 cases (37 per cent).

Acute and old thrombotic occlusions were present in 132 hearts (31 per cent). There was a total of 147 thrombotic occlusions. These hearts showed acute infarction in 35 instances (40 per cent). In a similar study,⁴ 75 per cent of the hearts having acute infarctions also had acute occlusions. The overall incidence of old infarction and focal fibrosis was 55.4 per cent while in 133 hearts (31 per cent) the myocardium showed no lesions at all. Two hundred eighty-six hearts (66.8 per cent) weighed 400 or more grams.

DISCUSSION

There is considerable difference of opinion between clinicians and pathologists,⁴ and even among pathologists,⁶ as to the incidence of acute thrombotic occlusions of the coronary arteries with or without myocardial infarction. Pathologic studies indicate that myocardial infarction follows acute coronary thrombosis in a high percentage of cases in clinical practice.^{7,8} However, there is no basis in forensic pathologic experience to justify the diagnosis usually made by the clinician, i.e., that the death, because it was sudden in an apparently healthy individual, resulted from an acute occlusion of a coronary artery. Fatal traumatic cases often reveal a degree of incidental coronary atherosclerosis much more severe than those in which the death is certified as due to coronary atherosclerosis.

Most persons dying suddenly and unexpectedly from coronary disease do not show acute changes in the coronary tree or myocardium. The morphology of the arteries is essentially the same as yesterday or the day

before. A mild exertional incident, i.e., walking, may be the same as the preceding day. The mechanism of sudden death in coronary artery disease appears to be physiological. This may be expressed simply as a disparity between the cardiac reserve at a given moment and the demands of a heart whose coronary arteries have previously undergone morphologic alterations. Cardiac reserve is dynamic in that it varies at any moment and may be dependent on qualitative as well as quantitative factors. If the reserve is not completely exceeded but is inadequate, sudden death may be delayed until infarction ensues.

In those cases in which an acute thrombotic occlusion is present, medical evidence is inconclusive as to the formation of the thrombus following an acute exertional episode at the time of death. Paterson⁹ has shown through detailed histologic examination that formation of coronary thrombi is a gradual process, therefore, activity at the time of death has no bearing on the inception of the thrombus. He believes that this does not exclude exertion or emotional stress as an etiologic factor in the formation of thrombi that are initiated by intimal hemorrhage caused by capillary rupture. Paterson suggests that physical or emotional stress, by causing temporary elevation in blood pressure and increase in coronary blood flow, may be factors in rupturing intimal capillaries. Texon¹⁰ has recently postulated a hemodynamic mechanism for intimal ulceration, thrombus formation, and intramural hemorrhage.

SUMMARY

A study of correlation between circumstances and sudden, unexpected death based on 428 consecutive autopsy cases of coronary heart disease is reviewed. In a fatal heart attack, the extent of physical activity cannot be correlated with the post-mortem findings that fail to reveal, in most instances, acute

changes of the coronary arteries or myocardium. Case histories are presented where emotional factors may be the predominant form of activity. The most frequently encountered lesion of the heart was progressive coronary atherosclerosis of varying segmental extent without thrombotic occlusion, occurring predominantly in middle-aged white males. The mechanism of sudden death in coronary artery disease appears to be physiological, affecting those hearts having previously undergone morphologic alteration of the vessels.

BIBLIOGRAPHY

1. *Census Tract Statistics*. New York, U.S. Census Bureau, 1950.
2. MASTER, A. M., DACK, S., and JAFFE, H. L. Relation of effort and trauma to acute coronary occlusion, *Indust. Med.* 9:359, 1940.
3. MASTER, A. M. Panel. *Coronary Thrombosis*, Medical Society of the County of New York, February 27, 1956.
4. RABSON, S. M., and HELPERN, M. Sudden and unexpected natural death, coronary artery sclerosis, *Am. Heart J.* 35:635, 1948.
5. BLUMGART, H. L., SCHLESINGER, M. J., and ZOLL, P. M. Angina pectoris, coronary failure and acute myocardial infarction, role of coronary occlusions and collateral circulation, *JAMA* 166:91, 1941.
6. EDWARDS, J. E. Pathologic spectrum of occlusive coronary arterial disease, *Lab. Invest.* 5:475, 1956.
7. FOORD, A. G. Embolism and thrombosis in coronary heart disease, *JAMA* 138:1009, 1948.
8. MILLER, R. D., BURCHELL, H. B., and EDWARDS, J. E. Myocardial infarction with and without acute coronary occlusion, pathologic study, *AMA Arch. Int. Med.* 88:597, 1951.
9. PATERSON, J. C. Relation of physical exertion and emotion to the precipitation of coronary thrombi, *JAMA* 122:895, 1939.
10. TEXON, M. A hemodynamic concept of atherosclerosis, with particular reference to coronary occlusion, *AMA Arch. Int. Med.* 99:418, 1957.

CHAPTER 34 THE RELATIVE ETIOLOGICAL IMPORTANCE OF VARIOUS STRESSES IN CARDIAC DISORDERS

William Dock

In the presentation of pertinent data to courts or referees in industrial liability cases, it is unusual to offer any facts whatever on the nonoccupational stresses in the patient's daily life. Indeed, one would gather from the testimony and decisions in such cases that the patient had experienced absolutely no stress except while on the job. The precipitation of illness or death is related only to the fact that there was a job that might have produced some stress. Two examples are now offered to show that this type of reasoning is often fallacious.

An electrician, two years after recovery from myocardial infarction, dropped dead at lunch, which had included two bottles of beer. The industrial examiner accepted the claim that death was due to the exertion of walking up a flight of stairs, an hour before lunch, and refused to consider that a stomach full of ice beer was a far worse stress and probably caused ventricular fibrillation. It was also known that the electrician played handball at least once a week, and had inter-

course with his wife at least four times each week, but it was the occupational flight of stairs, not these other and far greater stresses, which was accepted as fatal.

A clerk, after recovery from a very mild myocardial infarction, was encouraged by his doctor and permitted by his employer to resume his job. He walked to filing cabinets and moved folders, but did not lift heavy objects. When he died of a second episode that began at breakfast, the family physician testified and the referee agreed that his work hastened his death. The physician had not advised him to modify his diet, curtail his smoking, and forgo intercourse. He was still permitted to go pheasant hunting, but these stresses were ignored in making a judgment on the cause of death.

In the field of industrial law and medicine it is the rule that the occupational mote is observed, the nonoccupational beams are ignored. Or to use another simile, it is the occupational straw on the camel's back, not the heavy load of nonoccupational boulders,

which is blamed for the broken spine. As work becomes increasingly mechanized and the number of men who sweat at work steadily declines, it is obvious that the relative importance of factors outside the working hours will steadily increase, and physical stresses on the job will diminish.

Let us first consider the nature and severity of some common nonoccupational circulatory stresses and compare them with occupational ones. Coughing, or raising intra-abdominal pressure to hasten expulsion of urine, feces, or flatus impose burdens analogous to those caused by lifting heavy objects. These activities all entail sudden decreases and increases in cardiac filling and in peripheral resistance; they may cause vascular rupture or precipitate acute arrhythmias. Less severe but more sustained circulatory stress is imposed by sexual intercourse or masturbation. The effects of intercourse on heart rate were recorded by Boas several decades ago, and confirmed by Bartlett and Bohr last year.² Pulse rate is nearly doubled, cardiac output per beat is increased nearly 50 per cent, and systolic pressure rises about 30 per cent even in orgasms induced by masturbation, with minimal emotional or physical stress. This circulatory effect is comparable to that caused by running up two to three flights of stairs, while in intercourse the effect may be two or three times more severe or prolonged. These activities often precipitate acute pulmonary edema or angina in cardiacs who have no symptoms on the job.

Work in the home or garden, dancing, or participation in sports cause stresses equivalent to those of occupations that involve climbing stairs or ladders, hammering, or repeatedly lifting objects weighing 20 pounds or less. These activities may cause no alarming symptoms, but help set the stage for an attack of nocturnal dyspnea, or for gradually increasing edema and breathlessness. Going to or returning from work often provides the most intense physical effort and the most exasperating and frustrating experience of the day. Since the tax-gathering branch of government does not regard this activity as a necessary and deductible item, it would be

illogical for another governmental agency to include these daily stresses as occupational.

From the histories of patients with minor or severe dyspnea I have concluded that physical stresses due to the job exceed stresses only among the very small group of workers who need over 3500 calories to maintain weight; that the two causes are about equally important in jobs involving some manual labor; that off-the-job stresses predominate in light industry and professional services, and far outweigh occupational stresses in white collar groups. Of the off-the-job stresses, sexual activity is the main factor in young and middle-aged women, and in all other causes in men. Getting to and from work and moving household furnishings come very important factors after the decade

One of the most important nonoccupational stresses is imposed by nicotine. Cigarette smoking is very frequent, on and off the job, for all but heavy laborers, and the use of tobacco is common among hard workers whose jobs preclude smoking. In the presence of coronary disease, Davis and his co-workers at Johns Hopkins noted striking circulatory disturbance after smoking a cigarette in 60 per cent of the patients; Simon and colleagues report similar dysfunction to be the rule in tobacco chewers.² As the doberman's exercise test causes demonstrable dysfunction in fewer patients, it seems to say that chewing half an ounce of tobacco or smoking a pack of cigarettes cause much circulatory stress as climbing two flights of stairs. In addition, smoking often causes coughing paroxysms that further harass the heart.

Dietary factors, long ignored, are now recognized as serious causes of the acute episodes of heart failure and of coronary thrombosis. In the former, the history often shows unusually high salt intake preceded the midnight bout of dyspnea. A high-fat meal which shortens clotting time and increases blood viscosity, often is recorded in the histories of the events preceding acute myocardial infarction, especially when the clinical onset occurs between midnight and mid-

morning. Thus, the smoking and eating habits of workers deserve as much scrutiny as their physical activity when the causation of a heart attack is being analyzed.

Recent studies show that lack of physical activity is an important factor in raising blood cholesterol. Men on jobs involving hard work show lower rates and later onsets of coronary disease, and higher recovery rates from first attacks, than sedentary employees. In future, those who discuss work and heart trouble probably will spend more time on lack of physical activity than on excessive effort as an etiologic factor, and they certainly will spend more time on psychological stress than on physical stress. For there is abundant evidence that stimuli coming from the cerebral cortex, acting through hypothalamic centers and the sympathetic and endocrine systems, can alter heart rate, blood pressure, clotting time and plasma lipids. They thus can contribute to chronic or acute damage to the circulation.

But the weight of evidence is that the day's work is a negligible factor as a source of "emotional crises," or "unfavorable life situations," or of the "quiet desperation" which Thoreau regarded as the normal lot of man in nineteenth-century New England. Case histories in the psychosomatic literature and the novels about executives and laborers all show how important are the stresses arising from relations with parents, children, and the objects of intense sexual longing, and how unimportant are occupational stresses in causing unhappiness and frustration. One large, prosperous, and influential body of psychiatrists states that neuroses and psychoses result from a hidden struggle with the ghosts of infancy and childhood, and that the events of the present are relatively unimportant. They consider employment factors to be insignificant in comparison with home factors. Even those of us who think that too much emphasis may have been put on ancient, too little on recent psychic trauma, consider that in most cases it is not conflicts arising from work, but those due to competition with neighbors, conflicts in the home, and frustration of the intense human instincts for

gambling and for sexual adventure that are responsible for 90 per cent of the woes of the big executive and the little employee.

Our increasing knowledge of coronary disease has made it evident that physical work protects rather than damages the coronary arteries. Our observation of the rise in neurotic and psychotic disease, which seems to be associated with a rise in leisure time and reduction in working hours, makes us suspect that hard work actually protects the mind. Work diverts us from the petty or even from the serious problems arising in the home or in getting to or from work, or from not making as much money as one's wife thinks she must have to keep up with her relatives and friends. After studying occupational versus nonoccupational causes of emotional stress, I was reminded of the verses written by an American infantryman shortly before his death on the field of battle. Of life in the trenches he wrote: "There only, chastened by fatigue and toil, I knew what came the nearest to content / Discord and strife were what I used to know, / Heartaches, deception, murderous jealousy, / By War transported far from all of these, / Amid the clash of arms I was at peace." The office, the factory, the store, or the mine may provide moments almost as trying as the trenches, but the day's work is one of the most satisfactory parts of most men's lives, and retirement one of the most shocking experiences many men have to face. If we are to assign values to all the stresses, physical and mental, of all the employees who develop heart trouble, it is unlikely that occupational causes contribute 10 per cent of the total in the vast majority of cases where the industrial underwriter is charged with 100 per cent of the award for disability or death.

BIBLIOGRAPHY

1. BARTLETT, R. G., and BOHR, V. C. Physiologic responses during coitus in the human, *Fed. Proc.* 15 10, 1956.
2. SIMON, D. L., IGLAUER, A., BRADSTEIN, J. R., and RAKEL, R. E. Immediate effect of chewing tobacco on circulation of habitual chewers, *J.A.M.A.* 163 354, 1957.

REPORT OF THE PANEL ON PATHOLOGY

Jesse E. Edwards

Before I begin, in the name of the members of the panel on pathology I would like to thank Dr Belknap and his staff for the wonderful organization and planning of this Conference, which is being concluded today.

In our panel discussions we adopted the view that with regard to this particular Conference our concern was largely that of the testimony of the pathologist in the court and before Workmen's Compensation Boards. We recognize, of course, that in problems of heart disease and work there are many forms of heart disease that come into the picture.

In his presentation Dr Helpern showed us that patients who suffer acute attacks of heart disease at work obviously may have these on the basis of cardiac disorders other than coronary disease, including hypertension and valvular heart disease. In spite of this, we have concentrated our attention upon the problem of coronary heart disease, since it is the major concern in this area.

In the initial portion of the Conference we had presentations by Dr Hamilton and Dr Paterson concerning the findings on histologic examination of coronary arteries. These ob-

servations were used to support one or another theory of concern to atherogenesis. One theory is that mural thrombosis is the initiating cause in the formation of atheromatous disease, whereas another is that intimal hemorrhages occurring in the normal intima are ultimately the cause of atherosclerosis. In the discussion of these two causes, it was brought out that there have been propounded other causes for the development of atherosclerosis, including some peculiar alteration in the manner in which the vessel handles fatty substances. In the discussion of intimal hemorrhages, the view was broadened somewhat so that there was considered not only the role of intimal hemorrhages in atherogenesis, but also the possibility that in the established atheroma the intimal capillary by bleeding may give rise to complications. Dr Paterson felt that under stress dynamic circumstances may be set up whereby an intimal capillary hemorrhage may, in turn, do one of three things: (1) irritate the vessel wall producing spasm in that portion of the coronary system; (2) be so located that it increases the degree of the narrowing of the

vessel at that point; or (3) cause a fracture of the intimal surface and thus give rise to a complicating coronary thrombosis.

In his presentation and in the discussion which followed, Dr. Hamilton's view was that when thrombosis and intimal hemorrhage coexist the process probably works the other way. It is his opinion that the primary lesion is a thrombus and that because of it the nutrition to the intima containing the atheroma is cut off, resulting in infarction of the atheroma and the atheromatous intima. There is then secondary hemorrhage into the infarcted tissue. It was also brought out that, under certain circumstances of stress, perhaps an atheromatous plaque may be ruptured simply by mechanical causes and that such a rupture may be the focus upon which a complicating thrombus then develops.

Dr. Gould discussed unsuspected myocardial infarctions found in dying patients. To me this was a most important contribution and all the panel agreed. Several panelists supplied material in the discussion which confirmed Dr. Gould's experience that coronary atherosclerosis in the adult population in this country is a very common thing. Not only is it common to find atheromatous disease in the vessels, but there is the additional evidence of the presence of disease in the finding of healed myocardial infarction in patients who are admitted to the hospital for other reasons and who give no history of having had a myocardial infarction. In a smaller group of similar material concerning patients in whom electrocardiograms were done, healed myocardial infarcts were found subsequently at necropsy that were not uncovered previously. Therefore, we recognize that this material supports the view that certain established tests for coronary disease which we perform today do not necessarily uncover coronary disease when it is present, even to a significant degree. Obviously then, two persons who are being considered for employment, one with clear clinical history and evidence of myocardial infarction and one without it, may both have myocardial infarction in a healed state with co-existing severe coronary disease.

Next we had two papers from medico-legal sources, one of them from Dr. Spain and the other from Drs. Weinberg and Helpert. These were concerned with deaths that had come on suddenly and so were brought to the attention of the Medical Examiner's Office. In each of these studies an attempt was made to determine whether there was any predisposing factor in each of the cases of sudden death. While there were individual instances in which some emotional or physical episode of stress seemed to initiate the acute attack of myocardial ischemia, this was not the rule. In most instances the patient who died of a so-called acute coronary attack had been doing pretty much what he was accustomed to doing. Dr. Spain separated his cases into two major groups, those whose employment involved what we call physical labor, and those whose work was such that they tended to sit at a desk and did not exert themselves very much physically. There was a tendency for the patients who did a physical type of work to be older at the time of death from a sudden attack than those patients who were not engaged in a physical type of work when they died of their coronary disease.

At this point I would like to indicate that we feel it is important to stress to this group that there is something concerning a coronary attack which is very obvious to the pathologist but about which there tends to be a misconception among clinicians. It is our impression that the clinician often feels that the acute attack is synchronous with an acute coronary occlusion and that a thrombus which has occluded a vessel has brought on the attack. It was evident to the panel that there are many cases of coronary disease wherein patients have acute attacks and do not have actual acute coronary occlusions to coincide with the attacks. Whether or not an attack is associated with an acute occlusion varies depending upon what type of material the investigator studies. If one deals with patients hospitalized for acute myocardial infarction, about two thirds of those who die have an acute coronary occlusion and about one third do not. The fact that they have died

s for an automatic selection of cases. On the other hand, if one deals with sudden death brings the case to the attention of the local Examiner's Office, the result is quite diverse in that about three of the deaths are not associated with acute occlusion and one is associated with acute occlusion; in all cases the interval between the onset of symptoms and death is short.

It was brought out by Drs. Helpern, Hilton, Paterson, Weinberg, and Spain among patients who die acutely and have coronary thrombosis, many have histology that show histological evidence of an infarct older than the history of the acute attack. It appears that the situation is one in which a coronary thrombosis develops and gives no outward sign of it. Then, for some reason or other, some days later myocardial ischemia suddenly develops. Another point that we feel is important to stress is that among patients who die suddenly from coronary disease, whether or not they have an acute coronary occlusion (and I have already emphasized that many of them do not), most of them do not have an acute myocardial infarction. It seems, then, to be a situation in which the myocardium grows ischemic and develops either cardiac standstill or ventricular fibrillation in the absence of actual infarction of heart muscle.

There is one point concerning which we have a little disagreement. There is the problem of the pathologist who is asked to evaluate a certain circumstance in which the patient is subjected to some stress, either emotional or physical and an attack of pain occurs. Then after he seems to recover from the attack he dies suddenly three days later. At that time, if he has a myocardial infarction which is three days old, the pathologist tends to support the view that the initial episode, either emotional or physical, was responsible for his death. There may be a patient who is subjected to essentially the same stress and who has an attack of pain but grows better and has no more pain until he dies suddenly three days later.

If necropsy shows that he has no myocardial infarction, then the big question

arises, whether or not the initial episode three days before was or was not responsible for his sudden death when it occurred. This discussion led to a problem that was still unresolved at the close of our sessions. We have no way of telling how long an attack of myocardial ischemia that is short of producing myocardial necrosis will continue. We just do not know and I believe we really are unable to give an answer to this question. Our means of telling whether or not all the effects of acute ischemia that occurred three days earlier have vanished, or whether there is some subtle continuation of something during that period, are inadequate at this time.

That point was carried further by Dr. William Dock in the presentation in which he was concerned with occupational and non-occupational stresses in their relation to heart disease. He pointed out what had already been mentioned earlier here today, that the stresses of physical labor are obviously representative of only part of the story and that the workman has other stresses in his life and at home. If some stress is responsible for the precipitation of myocardial ischemia, how long does its effect last? This leads us to another type of problem. Suppose an individual has a serious fight with his wife one night and myocardial ischemia develops because of this stress, although he shows no outward signs of it. If he goes to work the next day and dies while he is at work, did the work kill him or was the argument the night before responsible? We do not really know the answer to this question. Since there is this great sphere of uncertainty when, as a group, we look back upon workmen's compensation awards, we feel that it is often just a matter of chance whether one family collects workmen's compensation benefits and another family does not. It seems to be simply fortuitous. In this regard, we have raised the consideration that there may be some purpose in supporting, as a group, a suggestion made by Dr. Dock. May it not be best to develop some type of broadened insurance coverage taking care of all cases in which death occurs while the individual is at work,

admitting that in some instances we just do not know whether or not the work is responsible? We would thereby avoid subsidizing the families of some patients dying of coronary disease simply because death occurred at work, whereas others are deprived of such benefits because death did not occur at work, although there may have been stress on the job that played a primary role in causation. We would not be in our present position of trying to separate, with our inadequate means, those cases in which the work is responsible and those in which it is not.

DR BELKNAP: Are there any other members of the panel on pathology who would like to supplement this report or anybody else who would like to comment?

DR JESSE E. EDWARDS: I am sorry to rise immediately, but I did not mention Dr Lev's studies on the conduction system, which represent a tremendous amount of basic work. In relation to the problem of coronary disease, we agreed with Dr Lev that we do not know just how far studies of the conduction system will lead toward any solution of understanding some of these problems. The question was whether, under certain circumstances, peculiarly localized lesions in vessels supplying the major conduction system might be responsible for cases of sudden death in coronary disease. It was the general feeling of the members of the panel that this probably *not* turn out to be the case, but these studies have to be done to learn the answer.

DR SYLVESTER E. GOULD: Would Dr Edwards review the proposals of Dr A. R. Moritz with regard to the giving of testimony?

DR EDWARDS: Dr Moritz made a plea for standardization of terminology and standardization of *perhaps situations*. With regard to standardization of terminology, I implied that on this general point we should realize that some patients have a thrombus and some do not. Some patients have an infarct and some do not. I believe we should be more specific in our terminology.

The other point was that he felt that in

order for testimony to be standard, that is, in the cases in which a heart attack is suspected of having been brought about by certain circumstances, it would be relatively simple to establish a certain series of cases by type, indicating those that would be logically considered to be connected with the event claimed to have caused the attack and those that one would class as having no connection.

Of course, this still leaves us with the problem that we cannot claim necessarily that a certain event did not cause an attack, even when we cannot see something in the heart muscle or anything new in the coronary arteries. It is possible that a certain situation can bring on an attack, whereas the pathologist can find nothing new. We cannot tell the difference, really, in a man's heart, whether or not he has had a recent argument with someone.

DR LEONARD GOLDWATER: Dr Spain's figures are derived from his experience in Westchester County. Having recently taken up residence in that particular county, I have more than an academic interest in the observation that, apparently, those who engage in physical types of work develop their coronary disease or their coronary deaths at a later time than those who engage in a sedentary or office type of work. I have made certain observations about the population in Westchester County and I believe that most of those who do engage in an office type of work sleep in Westchester County and work in New York City, whereas those who engage in the physical type of activity are more apt to live and work in the county. I do not know whether this is true, but I have this impression. I believe that there are other stresses associated with suburban living. Perhaps it is not just the office work but the fact that those people who work in offices must travel on a rather limited type of schedule between their home and their work every day, this, in itself, may be a factor causing earlier coronary disease than one would find in those who work and live in the same area.

DR ANGEL KEYS: Dr Edwards, are we to understand that the pathologists are now agreeing that, primarily, there are two basic

mechanisms to be chosen between in regard to the pathogenesis or the primary development of coronary heart disease? That we have (a) the possibility of intimal hemorrhage and (b) the possibility of mural thrombus and organization? Is that in essence most of the story or the whole story?

DR EDWARDS. There is the third possibility, which is yours. There was no agreement among the panelists as to the theories, the proper theories, concerning coronary atherogenesis. It was mentioned by a number of panelists that there is possibly something in the way in which the arterial intima handles fatty substances that may be related to the pathogenesis.

DR DAVID M SPAIN. I believe the discussion went a little further than that. There were people on the panel presenting papers that were concerned with only some of these local factors. I believe the discussion did bring out that there is a very broad point of view with a considerable body of evidence in the world that there are multiple environmental factors such as diet, the way we live, body types and activity, which may be the predominant thing in determining the rate of progression of coronary atherosclerosis. I believe the problem is how fast it will develop, which is not necessarily determined by any local hemodynamic factors. I believe this point should be emphasized.

DR MILTON HELPERN. There is just one point that I feel needs clarification after the question that Dr Keys asked, and that I thought Dr Edwards had made clear. I think our material in the medical examiner's office shows very clearly that the majority of people who die suddenly and unexpectedly from coronary artery disease die from a progressive atherosclerotic process, and I do not believe the moderator meant to imply that the lesions up to that point were associated with intimal hemorrhages. Some of them may have had hemorrhagic complications during the course of their development. I believe that as far as the intimal hemorrhages are concerned in these cases, they represent a complicating factor. Dr Paterson's material suggests that intimal hemorrhages are very

closely associated with the acute occlusions of the lumen. There is a difference of opinion regarding the formation of the acute thrombus, again, in our material, the acute thrombotic complications occur in only 25 per cent of the cases, whereas the great majority of the cases are simply occlusive processes brought about by gradually progressive atherosclerosis. Some of these show evidence of acute complications in the past, including occlusions of the lumen and mural hemorrhages.

DR. LOUIS N. KATZ. I believe we must be careful not to let the morphologists lead us to another defeatist attitude. Hemorrhages and thrombi may or may not be dealt with clinically, but every one of the instances has as its background atherosclerosis, and without going into particulars, I put it to this Conference that something may be done about this in the nature of diet.

DR. EDWARDS. It may have seemed that we have a defeatist attitude about this thing. Our opinion is the contrary. First of all, as a matter of background, I would like to refer again to the very common finding of old myocardial infarction. In Dr Gould's presentation he reported an incidence of 35 per cent of healed infarction that was entirely unsuspected, but he did not include in that material patients who had acute infarction, who also had healed infarction, but in whom the healed infarction was unknown. If we bring in these cases of healed infarction, it would raise the figure. Another point that should be mentioned with regard to healed infarction is that very often in these patients there are multiple scars representing many attacks associated with necrosis of muscle. Quite probably, in between these attacks there were many attacks of ischemia, which had the clinical form of angina that did not cause any necrosis of muscle. Although we have to start on this subject with people who have died, our feeling as pathologists is that, although coronary disease is a killer in one sense, on the other hand the individual can cope with coronary disease very well for a very long time and that the tendency for the individual patient with coronary disease is to live, not to die. The patient who dies with

acute myocardial infarction at this time is from a selected group for, although there is some variation in the reports of various observers, it may be said that, in general, only 15 per cent of patients who have clinical acute myocardial infarction die of their initial attack. Of those who die, over 60 per cent have pathologic evidence of previous myocardial infarction. They have had trouble before with which they have managed to live. Consequently in this regard we are not at all defeatists. We believe there is a great chance for patients with coronary disease to be active and useful for many years even after they have had clinical evidence of coronary disease.

The other point that we hoped would arouse Dr. Katz and others like him is this: One of the big problems, as far as sudden death in coronary disease is concerned, is that of sudden death, probably due to ventricular fibrillation or to cardiac standstill. Something happens to these people. Now, it is up to us as pathologists to throw it out to you. Your problem is to prevent sudden death in coronary disease. Try to figure out what happens to patients who have chronic coronary disease without any acute occlusion. They go about their normal way. They may be asleep, and they die suddenly. What is it that makes the myocardium fibrillate or what it is that makes it stop still? This is something for the physiologist to determine and for the clinician to prevent.

DR. J. C. PATERSON: Mr. Chairman, I think Dr. Edwards' report is a very true and admirable one. I believe that, perhaps, he exaggerated the importance that some of us placed on the etiology of atherosclerosis in our discussion. Certainly it was not mentioned in my written paper, although I did offer a suggestion in the discussion. This has been included in the summary of the moder-

ator and it has provoked Dr. Keys and Dr. Katz. Consequently I feel that I should give a very short explanation.

You have heard the statement that on this continent there are three types of students of atherosclerosis: (1) the group who believe implicitly in the lipid theory and who make up about 90 per cent of the whole; (2) the very small but vociferous group who are violently opposed to the lipid theory, and (3) the third group who do not know. I was a member of this third group until about a year and a half ago, when I changed my mind. I now am opposed to the lipid theory for two reasons, which are these: (1) I cannot find any significant association between the amount of lipid in the blood determined before death, up to four years before death, and the amount of lipid in the artery as examined after death, and (2) secondly, even in the early stages of the disease, I find evidence of material which I interpret as hemosiderin, which I attribute to intimal hemorrhage. This is my basis for changing to the Winternitz theory of atherosclerosis.

DR. SPAIN: I am not going to get involved in a controversy, but I feel that pathologists consider this problem from three points of view, in relation to work and physical activity. One is the effect of work and physical activity on the progression of the atherosclerosis; secondly, the possible aggravation of the progress of the underlying atherosclerosis or complicating factors, such as coronary thrombosis, by work or physical activity; finally, the effect of work on a person who has an *already damaged* myocardium as a result of the underlying atherosclerotic disease or a complicating development such as an acute coronary thrombosis. Those are three separate problems as far as work and activity are concerned and I believe they are our primary concern.

PART IV WORK CLASSIFICATION

CHAPTER 35 EXPERIENCE OF THE WORK CLASSIFICATION UNIT AT BELLEVUE HOSPITAL*

Lewis H. Bronstein

A Work Classification Unit should have primarily two important functions to serve. One is to follow the effect of employment on the course of heart disease as well as the effect of heart disease on employment records. The second is to determine its success in effecting employment of cardiacs, particularly those who are unemployed and have, on admission to the unit, no immediate job to which to return.

Our group of patients was completely unselected in that all patients who were referred were accepted. The referral source, age of patients, length of unemployment, occupational distribution, etiologic diagnosis, and functional and therapeutic classifications were noted on admission. These are correlated in this report with the following factors, which we think are the best indications for helping

to determine the functions mentioned in the previous paragraph. They are: (1) return for the visits necessary to complete the study, (2) whether or not heart disease is, in fact, present, (3) whether or not at least one return visit had been made one year later to determine the cardiac status and employment record.

Only the more significant tables will be presented, because of space limitation. The various tables relating the status at the one-year follow-up need a note of explanation at this time in order to prevent repetition during the ensuing discussion. The group labeled "Unknown" include those who never returned for subsequent follow-up, as well as those who returned at some time subsequent to the one-year cut-off time. Many of these were employed at the one-year period but are carried as unknown because they did not appear at the Unit at the stated time. Also, these tables do not supply the statistics on the switch of some employed on admission who

* Aided by a grant from the New York Heart Association. A presentation of patient selection of the Work Classification Unit of the Adult Cardiac Clinic, Third (New York University) Division, Bellevue Hospital, New York.

were unemployed one year later, because they are not ready at this time. They are not a large group and do not affect the conclusions materially. Subsequent reports will present this material. Those employed full time and part time will be counted as employed. All the other categories, including the unknown group, will be counted as unemployed. Such presentation weights the percentages against the employed group and should be borne in mind.

REFERRAL SOURCE

Table 35-1 shows that most of our patients came from the New York State Employment Service. This results in some selection since these people were collecting unemployment

insurance or were looking for jobs. They may have been better motivated than a group collecting funds from a public agency such as the Welfare Department. This latter agency is included in the group "Social Agency," which in its entirety represents only 10 per cent of our referrals.

It is to be noted that approximately 10 per cent of patients from the New York State Employment Service, Social Agency Group, and "Other" group did not complete the study and represent wasted effort. We do not believe that this is too excessive, particularly since the study includes the war years, 1942-1945, when great population shifts were taking place.

It also shows that almost 25 per cent of the entire group did not have heart disease. This

TABLE 35-1. PRESENCE OF HEART DISEASE AMONG PATIENTS IN RELATION TO REFERRAL SOURCE

	Total		New York State employment service		Division of vocational rehabilitation		Social* agency		Employer		Other†	
	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent
Presence of heart disease												
Heart disease	643	68	377	62	93	80	91	88	67	69	15	68
No heart disease	231	24	173	28	21	18	4	4	29	30	4	18
No diagnosis‡	78	8	64	10	2	2	8	8	1	1	3	14
Total	952	100	614	100	116	100	103	100	97	100	22	100

* "Social agency" includes hospitals, clinics, welfare, agencies, and New York Heart Association.

† "Other" includes self-referral and private physician.

‡ Patients were required to attend the Work Classification Unit two separate times for complete diagnostic work-up. Those with "No diagnosis" came for initial visit only.

TABLE 35-2. EMPLOYMENT STATUS OF PATIENTS IN RELATION TO REFERRAL SOURCE

	Total		New York State employment service		Division of vocational rehabilitation		Social agency		Employer		Other	
	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent
Employment status												
Unemployed	758	80	559	91	96	83	84	82	4	4	15	68
Employed*	194	20	55	9	20	17	19	18	93	96	7	32
Total	952	100	614	100	116	100	103	100	97	100	22	100

* "Employed" includes 13 students.

TABLE 35-3 PATIENTS WITH HEART DISEASE, STATUS ONE YEAR AFTER ADMISSION WITH RESPECT TO EMPLOYMENT IN RELATION TO REFERRAL SOURCE

	Total		New York State employment service		Division of vocational rehabilitation		Social agency		Employer		Other	
	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent
<i>One year status</i>												
Employed	251	39	136	36	32	34	25	28	56	84	2	13
Employed part time*	59	9	25	7	17	18	16	18	—	—	1	7
Unemployed	124	19	70	19	23	25	25	28	1	1	5	33
Out of labor market	16	3	3	1	3	3	5	5	4	6	1	7
Died	20	3	5	1	7	8	5	5	1	1	2	13
Unknown	173	27	138	36	11	12	15	16	5	8	4	27
Total	643	100	377	100	93	100	91	100	67	100	15	100

* Includes students

is in keeping with experiences of some other Units and is to be expected. Our previous publication¹ on part of this group shows that many of them had been told they had heart disease. The largest groups without heart disease came from the New York Employment Service and employer referral sources.

Table 35-2 shows the relation of employment status to referral sources. Eighty per cent of our patients were unemployed and, from that aspect, reflected a drain on community resources. As would be expected, the greatest percentage of unemployed was from the New York State Employment Service. The Division of Vocational Rehabilitation and the Social Agency group followed with the entire group average. The employer referral group, obviously, had a negligible group of unemployed.

Table 35-3 shows the status of those patients with heart disease, 643 of the original group of 952, at the first year's follow-up. It can be seen that the greatest deficiency in the follow-up occurred in those referred by the New York State Employment Service and by

TABLE 35-4 PATIENTS WITH HEART DISEASE, EMPLOYMENT STATUS ON ADMISSION AND ONE YEAR LATER IN RELATION TO REFERRAL SOURCE

	Admission		One year later	
	Em-ployed	Unem-ployed	Em-ployed	Unem-ployed
NYSES	30	347	161	216
DVR	16	77	49	44
Social agency	19	72	41	50
Employer	63	4	56	11
Other	3	12	3	12
Total	131	512	310	333

disease on admission and one year later, when correlated with the referral source. It will be noted that a substantial portion of these patients are now employed. The drop in the employer referral group can be seen as due to deaths and unknown status. It does not appear to be significant. A change from 131 employed and 512 unemployed at the time of admission to the Unit to 310 employed and 333 unemployed at the one year follow-up is significant. It must be noted again that some of those carried as not employed at this time interval were, in fact, employed but because they were not seen at the one year follow-up are carried as unknown and, therefore, unemployed.

at the one year follow-up

Table 35-4 presents a comparison of the employment status of 643 patients with heart

AGE

Our unit population was about evenly distributed in all age categories. These have been divided into 10-year groups as follows: Under 25, 25-34, 35-44, 45-54, and over 55. The first two age groups had 23 and 22 per cent of the entire population and the other three groups between 17 and 20 per cent.

A somewhat higher proportion of those in the first two age groups did not complete their diagnostic work-up—12 and 9 per cent as compared to the next three age groups—8, 5, and 4 per cent. This would indicate that the older age groups were more conscientious in completing their studies. A similar result was noted in the return for one year follow-up. However, it should be noted that the younger age groups would have been affected to a greater extent by the war years.

Of those who completed their work-up, there was a higher percentage of those with no heart disease in the youngest age group (37 per cent) and lowest in the oldest group (16 per cent). Those with heart disease were evenly distributed among all age levels. There was a slightly higher percentage of unemployed on admission in the two younger age groups than in the three older—about 85 per cent in the former and 75 per cent in the latter.

TABLE 35-5 PATIENTS WITH HEART DISEASE
EMPLOYMENT STATUS ON ADMISSION AND ONE
YEAR LATER IN RELATION TO AGE

	Admission		One Year Later	
	Em- ployed	Unem- ployed	Em- ployed	Unem- ployed
Under 25	19	100	63	56
25-34	21	115	62	74
35-44	32	78	63	47
45-54	28	112	62	78
55 and over	31	107	60	78
Total	131	512	310	333

Table 35-5 shows the relation between employment on admission and one year later in the various age categories. When one considers the difficulty of obtaining employment for older healthy people, it is indeed remark-

able that there was a 100 per cent increase in employment in the oldest age group.

LENGTH OF UNEMPLOYMENT

The length of unemployment was noted in 795 of the 952 patients. There was no relationship between this factor and failure to complete the study. It is to be noted that a very small percentage (3 per cent) of employed patients failed to complete the initial investigation. Ten per cent of the unemployed group failed to do so. There was no significant relationship between the presence of heart disease or the return for one year follow-up and the length of unemployment.

TABLE 35-6 PATIENTS WITH HEART DISEASE
LENGTH OF UNEMPLOYMENT ON ADMISSION AND
EMPLOYMENT STATUS AT ONE YEAR

Duration of of unemployment	Total	Employed at 1 year	Unemployed at 1 year
Less than 1 year	267	128	139
1 year	77	29	48
2-5 years	71	21	50
Over 5 years	14	1	13
Unknown	83	34	49
Total	512	213	299

Table 35-6 shows the relationship between this element and the employment status at one year for patients with heart disease. The column labeled "unknown for duration of unemployment" includes veterans recently discharged from military service, those who had recently graduated from school, and those for whom a work history had somehow not been recorded. It is to be noted that the shorter the period of unemployment, the greater the success in work placement.

OCCUPATIONAL DISTRIBUTION

The patients admitted to the Unit were classified as in Table 35-7. This is an abbreviated classification of that supplied by the National Office of Vital Statistics, Department of Health, Education, and Welfare (Feb. 20, 1956). It was modified to suit conditions in New York City with the help of Miss Margaret Barry, Vocational Counsellor.

to the New York Heart Association. The term laborers includes all unskilled labor.

The only categories that have sufficient patients in them are the following: (a) clerical, sales, and skilled, (b) semiskilled, (c) laborers. No relationship was found to exist between occupational classification and either return for completion of work-up or presence of heart disease. On admission, 30 per cent of the technical and administrative group were unemployed; 72 per cent of the clerical, sales, and skilled were unemployed; and 88 per cent of both the semiskilled and laborer groups were unemployed.

TABLE 35-7 OCCUPATIONAL CLASSIFICATION OF TOTAL GROUP ON ADMISSION

	Number	Percentage
Professional	5	1
Technical and administrative	36	4
Clerical, sales, and skilled	328	34
Semiskilled	278	29
Laborers	248	26
Students	13	1
No work history	44	5

Of the patients with heart disease, no difference was noted among the various classifications in regard to return for follow-up after one year. In regard to employment status after one year, a substantial increase in the proportion of patients employed was noted in each of the three largest categories. In the clerical, sales, skilled category, 53 per cent were employed one year later as compared to 28 per cent on admission. In the semiskilled group, it was 48 per cent as compared to 16 per cent, and in the laborer group, 39 per cent as compared to 10 per cent. We do not have as yet a breakdown as to the changes in occupational classification.

ETIOLOGIC DIAGNOSIS

Of the 643 patients with heart disease, Table 35-8 shows the relationship of employment on admission to the status of one year later. A definite increase in employment in each category is seen, although the congenital and "Other" groups are small.

There was no relationship between the

TABLE 35-8 PATIENTS WITH HEART DISEASE. EMPLOYMENT STATUS ON ADMISSION AND ONE YEAR LATER IN RELATION TO ETIOLOGY

	Admission		One year later	
	Em- ployed	Unem- ployed	Em- ployed	Unem- ployed
Congenital	7	20	11	16
Rheumatic	42	169	105	106
Hypertensive	6	39	17	28
Arteriosclerotic	33	118	76	75
Hypertensive and arteriosclerotic	13	56	27	42
Other	4	19	9	14
Possible and potential	26	91	65	52
Total	131	512	310	333

etioologic diagnosis and lack of faithfulness in returning for follow up. The percentages were similar for all groups.

FUNCTIONAL AND THERAPEUTIC CLASSIFICATION²

In this group, 117 patients rated as possible and potential cardiacs are omitted, leaving 526 patients. The group classed as I A had the poorest percentage of return at one

TABLE 35-9 PATIENTS WITH HEART DISEASE. EMPLOYMENT STATUS ON ADMISSION AND ONE YEAR LATER IN RELATION TO FUNCTIONAL AND THERAPEUTIC CLASSIFICATION

	Admission		One year later	
	Em- ployed	Unem- ployed	Em- ployed	Unem- ployed
I A	3	35	16	22
I B	29	91	62	58
II B	33	125	74	84
II C	30	114	77	67
III C	10	52	16	46
IV	—	4	—	4
Total	105	421	245	281

year. This may well be due to the fact that these patients felt so well that they saw no reason for returning.

Table 35-9 relates the employment status on admission and one year later of the patients in the various groups in this category.

There was a definite increase in employment for all cardiacs whose classification was better than III C. This group did not show much difference. The Group IV patients were obviously unemployable.

DISCUSSION

It can thus be seen that with as completely unselected a group as possible, there are very few categories of patients who were unsatisfactory from the various standpoints mentioned at the start of this report. It must be realized that there are certain factors in our social structure that influenced our results. World War II which, in a way, was responsible for the initiation of the Work Classification idea, also hindered a more satisfactory study. This was due to the displacement of population by induction into the service and removal to more favorable employment markets.

The generally tight labor market during the past 15 years helped our statistics in providing more employment opportunities for handicapped people. In a different type of market, the employment results would undoubtedly not have been as good.

The Unit operated without a social worker or vocational counselor. A report of physical capacity based on clinical opinion was sent to the referring agency. In addition, a great deal of time was spent with each patient, outlining to him the types of jobs we felt would be suitable. This was as far as we could go with limited personnel. When it is remembered that the unemployed group had no job to which to return, it is wonderful that so many became employed.

We feel that, except for the functional and therapeutic group which is worse than III C and the group unemployed over five years, there should be no restriction on the type of patient admitted to a Work Classification Unit. The funds will be very well spent.

BIBLIOGRAPHY

- 1 GOLDWATER, L. J., BRONSTEIN, L. H., and KRESKY, B. Study of 175 "cardiacs" without heart disease, *J. I.M.A.* 148 89, 1952.
- 2 NEW YORK HEART ASSOCIATION, INC.: *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Blood Vessels*, by Criteria Committee, HAROLD E. B. PARDEE, Chairman. 5th ed. New York, Am Heart Assn, 1953.

CHAPTER 36 EXPERIENCE OF THE CARDIAC WORK CLASSIFICATION UNIT IN BOSTON, MASSACHUSETTS*

Richard J. Clark

BACKGROUND AND METHODS

The Cardiac Work Classification Unit in Boston, sponsored through the co-operation of the Massachusetts Heart Association, the Bay State Society for the Crippled and Handicapped, and the Division of Chronic Diseases of the Massachusetts Department of Public Health, started operation in October, 1952. As has been true with many of these units, the early phase was slow, with sessions held about every two weeks. Gradually referrals increased to the point where full sessions are now held weekly, except during the summer months, with regular evaluation of four new patients. This year appointments have been booked one to two months in ad-

vance. New patients seen each season have increased from 41, 70, 80, and 114 to a probable 140 this year. From 4 to 6 follow-up patients are seen at each session.

It was the decision from the first that this unit would operate primarily on clinical grounds, using the equipment of a regular cardiac clinic with the personnel and general methods of operation of a work classification unit. While granting the desirability of associated special research in the field of exercise tolerance testing and the like, our unit had neither space nor personnel for this. Beyond this it was thought that we should learn what could be accomplished with ordinary measures so that our methods and results might more readily be transferred to other existing clinics.

Procedures carried out routinely have included electrocardiograms, chest x-rays and fluoroscopy, serology, sedimentation rate, hemoglobin, and urinalysis. Exercise toler-

ance tests (Masters) have been carried out only as an aid to diagnosis in certain questionable cases. Patients are frequently observed at stair-climbing and walking as a part of the complete clinical examination. Special psychological, vocational, and aptitude testing has been carried out in indicated cases outside of the unit through our vocational associates, the J.O.B. (Just One Break) Program.

Unit personnel has included three regular physicians (with other visiting and temporary physicians), a half-time social worker; a part-time vocational counselor (and at times two), first from the State Division of Vocational Rehabilitation and more recently from the J.O.B. Program, a laboratory and electrocardiographic technician for unit sessions; and most important, a full-time secretary.

As a corollary to our evaluation program, the unit in the past two years has attempted indirectly to carry out industrial placement in appropriate cases through the J.O.B. Program, working under a grant from the Massachusetts Heart Association. This activity was prompted by the fact that in our first two years of experience, existing agencies were doing very little toward placing the patients whom we had evaluated. In our first year of experience with J.O.B. we found 26 patients at work out of 46 *definitely employables* referred, contrasted with 47 similar referrals elsewhere with 5 placements. This alone has demonstrated to us that with the proper approach jobs can be found for cardiacs.

PRESENT STUDY

It has been our desire to attempt the development of profiles of employable and unemployable cardiacs in industry. Accordingly, through the co-operation of the statistical department of the State Department of Public Health an analysis of findings to date has been attempted. In our experience about 70 per cent of the patients involved have had degenerative heart disease. It is this group which poses the largest problem to industry.

For this reason, the major emphasis of the study has been placed here.

Detailed studies are limited to patients evaluated through June, 1956, only, in an attempt to have reasonable follow-ups. Unfortunately, dealing with a section of cardiac patients of this type, there is a definite number for whom satisfactory follow-up is not possible. We regret that this reduces the numbers in subgroups to a point, in some instances, where the figures are not significant and must be viewed chiefly as trends or indications.

This study has also pointed up very clearly to us the necessity for developing specific and complete record forms to include routinely many factors that the neophyte in this field might not consider significant. The desirability of coding on punch cards from the start is clear.

CRITERIA OF EMPLOYABILITY

Throughout this study patients are listed as *Employable*, *Questionably Employable*, and *Unemployable*. These represent the combined judgment of the physicians and vocational and social workers, either at the group conference following evaluation or in some cases after the vocational group has carried out more extensive testing. This evaluation is not based upon cardiac capacity alone, but upon this, plus factors of personality, motivation, age, vocational history, aptitudes, and skills, all of which enter into employability in the regular labor market. Those listed as *questionably employable* are those whose prospects in the regular labor market we consider highly dubious but for whom special effort might be made. The *unemployables* are those on whom we do not believe it worthwhile or wise to expend the effort to attempt placement.

However, our *functional* and *therapeutic classifications* are judged strictly on the basis of cardiac status.

OVER-ALL UNIT STATISTICS

Certain of the facts of general unit statistics are presented in Table 36-1. In part they

may be peculiar to our own unit and present chiefly a background for later material

They indicate a real interest on the part of the family physician for help in this area, but it may be added that in many instances the

TABLE 36-1 CARDIAC WORK CLASSIFICATION
UNIT: GENERAL UNIT STATISTICS
(October, 1952, through January, 1957)

1	Total patients evaluated, 377	
2	Sources of referral	
	Family physician	176
	Industries	38
	Insurance clinics	14
	Hospital clinics	69
	Public agencies	80
3	Diagnostic categories	
	Coronary, arteriosclerotic, or hypertensive heart disease	261
	Rheumatic heart disease	88
	Congenital heart disease	7
	Normal hearts	18
	Other	3
4	Age ranges	
	10 to 20 years	10
	21 to 30 years	25
	31 to 40 years	29
	41 to 50 years	103
	51 to 60 years	159
	Over 60	51
5	Sex Females, 38, Males, 339	
6	Number evaluated as <i>employable in the labor market</i> 235 or 60% of 377 patients through January, 1957	
7	Lack of complete follow-up of all groups prevents exact total employment figures, but the following were essentially up to date through June, 1956	
	Patients evaluated	314
	Deemed employable in labor market	185 or 59%
	Number known as employed	116 or 63% of <i>Employables</i>
	Of these	
	Returned to job at evaluation	38
	Returned to previous job	20
	New employment	47
	Returned to prior job with adjustment	11
	Total	116

request for referral originated with the patients. A need for assistance by public welfare agencies in determining possibilities for rehabilitation among their applicants is evident.

As already mentioned, degenerative heart disease plays the major role of problems pre-

sented to us, 261 of 377 patients. Rheumatics are about a third as numerous with 88, but still a significant group. The small number of normal hearts, 18, is noteworthy.

The very major age load has been in the 6th decade, with 159 patients, which is understandable in view of the increasing difficulties of job finding for those in this period, regardless of vascular disease. The next highest group, 103 in the fifth decade, represents an overlap of degenerative and rheumatic heart disease. The significant group of 51 in the sixties presents a most difficult employment problem on the basis of age alone.

The presence of an overwhelming majority of males, 339 to 38 females, is in part explained through the fact that we have not evaluated women for work as *housewives* as has been done by some units.

Of the entire group we have deemed approximately 60 per cent as *employable* and of these some 63 per cent have been employed.

RHEUMATIC HEART DISEASE AND CONGENITAL HEART DISEASE

To date no detailed studies have been completed with the relatively small group of patients in these categories. Forty-eight or 70 per cent of the 69 rheumatic heart patients studied were evaluated as definitely employable and of these 32, or 67 per cent of the *employables*, were employed. This is slightly above the general clinic average and reflects in part a relatively lower age range, and somewhat less hesitation on the part of informed employers to hire rheumatics than those with degenerative disease. The unemployables were chiefly patients in the later stages of the disease process with congestive failure and a poor outlook, but in a few cases represented serious personality or psychological problems.

The congenital group consisted of seven patients, all of whom had significant defects. Four have been satisfactorily employed, three at new work. Their employment problems in general are similar to those of the rheumatics, whom they tend to parallel in age.

HYPERTENSIVE DISEASE

In our breakdown of categories for study, we separated those with myocardial infarction and those with angina pectoris who had not had infarction. Both of these groups include an appreciable number with hypertension. We were surprised to find a residual of only 18 patients with primary hypertension and hypertensive heart disease, a small number from which no conclusions can be drawn. Eight patients of this group were evaluated as employable and, of these, four were returned to their prior employment, none found new employment. Three of the four have continued well at work for over three years, while one has discontinued working.

It is our opinion that the patients with severe hypertension, especially those with sustained diastolic pressures of 120 mm Hg and over, constitute a highly questionable group for employment. On the other hand, there are many with uncomplicated and relatively asymptomatic, mild hypertension who are capable of all types of work for long periods of time. Detailed statistic studies of this group are needed.

CORONARY HEART DISEASE

Angina Pectoris Without Known Myocardial Infarction

Again we regret the relatively small number, 44 patients, in this category. The largest age group was in the 6th decade with 19 patients, and the next largest group of 14 cases was in the age range 60 to 69 years. This fact reduces employment prospects sharply. There were 23 patients who were deemed employable, 52 per cent of those evaluated.

Twenty-seven patients had adequate follow-up; of these 18 were employed, 16 on a permanent full-time basis, 78 per cent of *definitely employables* found work; 8 of these, new employment.

Over a period of 6 to 48 months, 17 of the 18 employed patients have continued at work, with three finding re-employment. There has been only one death in this group,

accounting for the one patient not continuing on the job, and this death was nonindustrial. Apart from this one patient, none have deteriorated physically and 3 have improved, the latter being patients in whom frequency of angina definitely decreased after employment.

The presence of angina pectoris in itself is no bar to employment. Anginal patients with reasonable cardiac function and with other qualifications to render them employable may be employed and continue employment over significant periods of time without deleterious effect. We have observed definite improvement of angina under conditions of work which we would interpret as due to relaxation of worry and family tension.

Patients Who Have Had Myocardial Infarction

General Statistics

The patients in this study are limited to those in whom a definite diagnosis of myocardial infarction has been substantiated either by our own electrocardiograms or by previous tracings. Any questionable cases were eliminated. This leaves us with 141 patients initially evaluated through June, 1956, and 102 patients with satisfactory follow-up.

General statistics for this category are enumerated in Table 36-2.

It was our evaluation that 87, or 62 per cent, were definitely employable. Of this number 62 (70 per cent) found employment, 35 were placed at new employment. We consider this a victory in this cardiac group, which is considered by some as essentially unemployable as far as new work is concerned, and certainly a refutation of any such hypothesis.

The reasons for *unemployability* are of some interest. They point up clearly that severity of heart disease alone is not the only deterrent to work. Age is an appreciable factor. Especially with present-day fringe benefits, the very much more important factor is the patient's ability to perform the work. For those over 65 (of which there are a number in the group) regular employment is largely a matter of lucky chance. Psychologi-

TABLE 36-2 GENERAL STATISTICS. MYOCARDIAL INFARCTION CASES

1. Patients evaluated October, 1952, through June, 1956, 141	
2. Number with satisfactory follow-up data, 102	
3. Age ranges	
20 to 29 years	2
30 to 39 years	8
40 to 49 years	37
50 to 59 years	71
Over 60	23
4. Sex: Males, 134, Females, 7	
5. Following study evaluations for employment were as follows	
Definitely employable	87 (62 per cent)
Questionably employable	21
Unemployable	33
6. Reasons for unemployability were listed as follows	
Heart disease too severe, 6	
Severe heart disease plus age factor, 11	
Severe heart disease plus important psychological defect, 6	
Age primary factor, 3	
Personality and psychological factor, 5	
Other defects, 2	
7. Status at time of evaluation	
Working	22
On sick leave	11
Unemployed	108
8. Subsequent status of 102 patients with satisfactory follow-ups	
Returned to or found permanent full-time employment	56
Permanent part-time employment	4
Temporary full-time work	1
Placed in sheltered workshop	1
Employed	62
70 per cent of <i>Definitely Employable Group</i> were employed	
Returned to prior job without work adjustment	13
prior job with work adjustment	12
prior employer, new work	2
Placed at new employment	35
Employed	62
40 per cent of <i>Definitely Employable Group</i> found new employment	
Unemployed	40

cal factors include severe neurosis, marked lack of motivation, alcoholism, as well as bad personality traits, all judged to be of an insurmountable degree, these are obviously significant in number

We have been impressed by the willingness of many employers, not only to take back old patients, but to co-operate in making recommended work adjustments. The 22 patients working at referral were sent in chiefly for evaluation of their capacity to continue a given job. The 11 on sick leave were convalescent recent infarctions.

We have also been impressed by the fact that most patients capable of work have been capable of full-time employment. In a few instances relatively recent infarction cases have been recommended for half-time work the first month, but they have all graduated to full-time work.

Follow-up of Employed Group

Table 36-3 presents the status of the employed group subsequent to evaluation.

The method of new job-finding is of some interest. Here, as in other categories, we have often found it best to direct the patient to go out and find his own job. Frequently specific suggestions as to possible openings and avenues of approach have been made by our counselors. Of 35 new jobs found, 22 were by the patient himself, 9 by private agency (chiefly J.O.B.) and 2 through a state agency.

The type of new employment has been for the most part in light manufacturing, notably in the growing electronics industry, which can offer the cardiac ideal types of bench assembly work provided he has the needed hand and eye dexterity. Service jobs of various types and sales work came next on the list.

We were interested in learning how soon patients returned to work after their infarction. Only 11 of the 60 patients returned in less than 4 months, while the great majority of 43 went back after 6 months. This reflects two facts. In the first place we are certain that many family physicians are overconservative and overly restrict their patients after infarction. Secondly, we received many patients who had been seeking employment unsuccessfully on their own for some period of time before evaluation.

The follow-up of those continuing satisfactorily with employment is most encourag-

TABLE 36-3. MYOCARDIAL INFARCTION CASES.
STATUS OF EMPLOYED GROUP

1. Patients with satisfactory follow-up ranging from 3 to 48 months, 60	
At new employment, 35	
2 Method of new job finding 35 patients	
On own	22 (often with advice from unit or other counselors)
Private agencies	9
State agency	2
Through old employer	2
3 New employment found 35 patients	Job classification, 35
Manufacturing—light	14
Services	9
Sales	7
Office	3
Construction	1
Transportation	1
4 Time from most recent infarction to start of work, 60 patients	
Under 2 months	2
Under 3 months	3
Under 4 months	6
Under 5 months	3
Under 6 months	3
Over 6 months	43
5 Number who have stopped first employment after evaluation, 16	
Reasons	Job ceased 6
Deaths	5 (None at work)
Noncardiac illness	2
For better job	1
Moved from area	1
Family sickness	1
Cardiac deterioration	0 (other than deaths)
Job too strenuous	0
Number finding and continuing subsequent re-employment, 6	
6 Number presently continuing with employment, 50 (83 per cent)	
7 Medical follow-up, most recent (range 3 to 48 months)	
Comparison with original evaluation	
Physical status	Unchanged 37
	Improved 3
	Deteriorated 3
	Died 5
	Uncertain 12 (Not due for first medical follow-up or no check in 1 year)

TABLE 36-3. MYOCARDIAL INFARCTION CASES
STATUS OF EMPLOYED GROUP (Continued)

Psychological status:	Unchanged 37
	Improved 6
	Deteriorated 0
	Died 5
	Uncertain 12
8. Deaths in the group	
Case 1 M. S. Age 54 Class II C. Shoe worker	
Returned to old job, heavy work, against our advice, one year after evaluation and 13 months after initial infarction. Died suddenly two days after start of work. Had not followed medical advice. Could have been work-induced.	
Case 2 J. W. Age 56 Class II B. Chief of Town Fire Department. Continued prior work.	
Died 1½ years after evaluation and 2 years after initial infarct of recurrent myocardial infarction. Onset not in relation to work.	
Case 3 J. H. Age 47 Class II B. Gasoline station operator. Developed phlebitis and died of pulmonary infarction after one month of hospitalization, one year after evaluation, and 2 years after original infarction. Onset not work-related.	
Case 4 G. W. Age 43 Class II B. Clerical worker	
Died following chest p in starting while en route to work, 16 months after evaluation. Had 2 prior infarcts 3 years and 1½ years before death. No probable work relationship.	
Case 5 L. E. Age 51 Class II B. Tool worker	
Developed gradually increasing cardiac disability and had given up regular work. Died 14 months after evaluation and 23 months after infarction. Not working at time of death. Any work relationship doubtful.	

ing and would tend to indicate successful selection and placement. As indicated, 50 of the original 60 employed are continuing work. There have been 6 finding re-employment for one reason or another. It may be noted that none found his work too arduous and none was required to stop work for cardiac deterioration except in 5 cases that died.

Medical follow-up indicates that the physical status (based on history, examination and at times on repeat electrocardiogram) was unchanged in the great majority (37), improved in 3, and deteriorated in 3, but not to a degree requiring cessation of work. The psychological outlook was noted as im-

proved in 6 instances, but this must be a conservative figure as a most common remark has been, "You don't know how much better I have felt since getting busy."

Deaths

There have been five deaths in the employed group (Table 36-3). Of these only one would appear to have any work relationship, and this was in a man starting a job against advice.

Comparison Between Employed and Unemployed Patients. Discussion

Table 36-4 shows a comparison of various factors listed under Group I, *Employed*, Group II, *Deemed employable but not yet employed*, and Group III, *Deemed unemployable or questionably employable and not employed*. Since all these patients have been evaluated more than 6 months past, it is likely that the majority in Group II may continue as unemployed. While there are 102 cases analyzed, it is fully realized that subgroups are still so small as to make percentage figures of questionable statistical significance in some instances. Various factors will be commented upon.

AGE As might be expected, Groups II and III have a higher proportion of those over 60 and Group III has a higher proportion in the sixth decade. We clearly recognize that age over 60 makes employment more difficult.

EDUCATION The definitely striking factor in these figures is the marked increase of special or vocational training in the employed group and also the increased percentage of high school graduates in the employed group. Higher education would appear associated with greater facility of job-finding.

PREVIOUS WORK EXPERIENCE* We are especially struck by the preponderance of unskilled workers in Group II, which may be one of the reasons for unemployment while deemed employable. On the other hand, we

see Group I composed chiefly of semiskilled and skilled workers. It may be judged that the high proportion of semiskilled and skilled workers in Group III is coincidence and certainly not the reason for unemployment. It is well recognized by our vocational counselor that increase in degree of work skill facilitates employment. The severe cardiac who has never done anything but unskilled labor is one of the most difficult placement problems.

FINANCIAL ASSISTANCE These figures are too meager to be of special significance. The more severe cardiacs, and less employable, are more apt to be on public assistance. The relatively high percentage of Group II on assistance might suggest this as a possible factor deterring them from work. It is significant that 12 per cent of Group I were on assistance but in spite of this they became employed. It would be unwise to draw any conclusions regarding compensation. The fact remains that while assistance or compensation may prove a deterrent to work in some cases, the more ambitious individuals with family responsibility are not satisfied with the lot it offers, and strive to obtain employment.

TIME OF LAST WORK TO EVALUATION. It is our strong belief that the sooner a cardiac can get back to work the better, and the longer he waits the less good are his chances of employment. These figures show no consistent trend to prove this point, but are suggestive when we note that the largest proportion of those waiting over 2 years were in Group III, while the time interval decreased progressively in Group I.

MOTIVATION As would be expected, good motivation is clearly higher in the employed group while poor motivation is greatest in Group III. It may well be significant here that questionable motivation is highest in Group II. While these figures do not clearly define the situation, there is no question that motivation of the individual is of prime importance in employment.

CARDIAC ANXIETY We observe a clear preponderance of marked cardiac anxiety in Group III, those unemployed, but the figures otherwise are not significantly different.

* Unskilled is defined as work requiring no special training or intelligence beyond average to perform. Semiskilled is that type of work requiring some on-the-job training of a few weeks' duration and requiring average or better intelligence. Skilled work requires training of a year or more and superior intelligence.

TABLE 36-4. MYOCARDIAL INFARCTION CASES: 102 CASES WITH SATISFACTORY FOLLOW-UP

		Factors in comparison of employed and unemployed patients					
		Group I ^a (60)		Group II ^a (22)		Group III ^a (20)	
Factor number, total		Num- ber	Per cent	Num- ber	Per cent	Num- ber	Per cent
1	Ages						
	Under 40	5	8†	3	14	1	5
	40 to 49	24	40	6	28	3	15
	50 to 59	26	43	10	45	12	60
	Over 60	5	8	3	14	4	20
2	Education						
	8 years or less	14	25	7	33	8	44
	Attended high school	24	43	13	62	7	39
	High school graduate	14	25	1	5	3	17
	Attended college	4	7	0		0	
	Not recorded	4		1		2	
	Additional special or voca- tional training	18	32	1	5	0	
3	Previous work experience						
	Unskilled	10	17	12	55	3	15
	Semiskilled	26	43	3	14	8	40
	Skilled	19	32	5	23	9	45
	Professional	5	8	2	9		
4	Financial aid at evaluation						
	On public assistance	7	12	8	36	7	35
	On workmen's compensation	3	5	2	9	3	15
	Compensation pending	4	7	0		2	10
5	Working at evaluation	19	32	1	4	5	0
6	Time from last work to evalua- tion						
	Under 6 months	17	28	9	41	2	10
	6 to 12 months	14	23	5	23	8	40
	1 to 2 years	8	13	4	18	4	20
	Over 2 years	2	3	3	14	6	30
7	Motivation						
	Good	53	88	13	59	11	55
	Questionable	7	12	7	32	4	20
	Poor	0		2	9	5	25
8	Cardiac anxiety						
	Marked	7	12	3	14	6	30
	Moderate	16	27	7	32	2	10
	Minimal or none	37	61	12	55	12	60
9	Classifications						
	I	10	17	5	23	0	
	Functional II	47	78	15	69	16	80
	III	3	5	2	9	4	20
	IV	0		0		0	
	Therapeutic A	0		0		0	
	B	42	70	13	59	7	35
	C	18	30	9	41	13	65
	D	0		0		0	
10	Number of prior infarcts						
	1	52	87	19	86	15	75
	2	7	12	3	14	3	15
	3	0		0		2	10
	4	1	1	0		0	
11	History of angina before infarct	13	22	8	36	6	30
12	Angina after infarct	31	51	10	45	14	70
13	Hypertension at evaluation	22	37	8	36	7	35
						2	10
14	Cardiac enlargement at evalua- tion by x-ray						
	Marked	1	1	0		6	30
	Moderate	11	18	2	9	7	35
	Minimal	16	27	10	45	5	25
	None	32	53	10	45		

TABLE 36-4 MYOCARDIAL INFARCTION CASES 102 CASES WITH SATISFACTORY FOLLOW-UP (Continued)

Factor number, total		Factors in comparison of employed and unemployed patients					
		Group I* (60)		Group II* (22)		Group III (20)	
		Num- ber	Per cent	Num- ber	Per cent	Num- ber	Per cent
15	History of previous congestive failure	7	12	3	14	5	25
16	On digitalis	11	18	2	9	5	25
17	Mercurial diuretics	2	3	1	4.5	4	20
18	Obesity present	17	28	6	28	4	20
19	ECG findings†						
	Normal	6	10	1	4.5	0	
	Posterior infarct pattern	25	42	4	18	7	35
	Anterior infarct pattern	17	28	11	50	9	45
	Combined patterns	2	3	1	4.5	1	5
	Aneurysmal pattern	0		0		1	5
	RBBB	1	1.7	2	9	0	
	L V H pattern	7	12	3	14	4	20
	Other ST or T changes	6	10	1	4.5	0	
20	Subsequent death	5	8.3	1	4.5	6	30

* Group I Employed group, full-time employment

* Group II Deemed employable but not as yet employed

* Group III Deemed unemployable or questionably employable and not employed

† Calculated to nearest percentage point

‡ In some cases two factors recorded for single patient. Percentage is that of total group showing the factor

While cardiac anxiety unquestionably serves as a deterrent to work, it is one susceptible of improvement and in most cases is not a long-term bar to employment.

CARDIAC CLASSIFICATION, IN ACCORDANCE WITH AMERICAN HEART ASSOCIATION. It must be admitted that these classifications tend to be arbitrary and may well vary from unit to unit. Probably those shown tend to be graded a bit higher than might have been the case elsewhere, yet they should be comparable between the groups. Of particular interest is the relatively high percentage of Class I patients in Group II, which in conjunction with Factors 3 and 7 especially suggest that it is not the heart disease itself that has deterred these patients from employment. An almost equal preponderance of all groups were classed as II. Those of Class III are more numerous in the unemployed group and smallest in the employed group. It is suggested that the therapeutic classification may be of more importance in the employment

picture with the clear preponderance of Class C patients in the unemployed group and a minority in the employed group. Again the high percentage of Class B in Group II points out that cardiac capacity in itself is not of major importance in the unemployment of this group.

NUMBER OF PRIOR INFARCTS. From these figures it would appear that the number of prior infarcts alone plays relatively little part in employability, which conclusion is in agreement with our clinical impression.

OCCURRENCE OF ANGINA PECTORIS. The history of angina pectoris before myocardial infarction (and this applies to long-term angina, not prodromal symptomatology) is relatively consistent between the groups, being a little lower only in the employed group. Again the presence of angina after infarction is not strikingly different except for some increase in the unemployed group. It is arresting to note the over-all frequency of angina in this entire series of cases, slightly

over 50 per cent. This corresponds with our finding noted under angina pectoris, namely that this symptom complex is by no means a bar to employment.

HYPERTENSION Again we are surprised to see the consistent frequency of 36 per cent showing this finding in all three groups. For statistical purposes, hypertension was defined as a diastolic pressure of 100 mm. Hg or more. A further breakdown of pressure levels might be more informative. It appears that the complicating presence of hypertension in itself plays little part in employability, but in our opinion those with diastolic levels of over 120 mm. Hg are usually not good employment prospects.

CARDIAC ENLARGEMENT Here the finding of a preponderance of marked and moderate enlargement among the Group III infarction cases is in accordance with our clinical judgment that this is a relatively poor prognostic factor, but certainly not an absolute one. Here again the relatively high percentage of Group II without enlargement goes along with the contention that the cardiac state in itself is not the chief reason for nonemployment of this group.

HISTORY OF CONGESTIVE FAILURE This factor includes failure at the time of infarction or subsequently. It would have been well to have separated these. There is a higher incidence in Group III, but the difference is not as striking as might have been anticipated.

USE OF DIGITALIS This factor is probably not of great significance, since it depends very much upon the philosophy of the family doctor. There is an insignificant difference between groups.

MERCURIAL DIURETICS This indicates that the patient has been on regular mercurial diuretics as a part of his regime. Obviously the cases are too few to be significant, but use is more common in Group III. Use in two employed patients is noteworthy.

OBESITY The presence of obesity is surprisingly well distributed through the groups, actually being a bit more common among the employed patients.

ELECTROCARDIOGRAPHIC PATTERNS Two

conclusions may be drawn. First, there is a slight predominance of normal tracings exhibited by the employed group. Second, various other findings and patterns are fairly evenly distributed among the groups, tending to confirm our opinion that the electrocardiogram is useful only in helping to confirm or establish a diagnosis.

SUBSEQUENT DEATHS. The higher proportion in Group III is to be expected and tends to validate the classifications. Again the small percentage in Group II continues to argue against serious heart disease as a cause of their unemployment.

Conclusions Regarding Comparative Factors Influencing Employment in Myocardial Infarction Cases

1. Age is a deterrent to employment. Persons over 60 years of age as well as those in the latter part of the sixth decade present greater problems.

2. Higher education, particularly special vocational training, facilitates employment.

3. While not conclusively demonstrated, it is our opinion that the higher the degree of work skill, the greater the likelihood of employment.

4. No definite conclusion can be drawn as to the influence of public financial aid or compensation upon employability.

5. The shorter the time interval from work incapacity to evaluation for work, the greater the chance of employability.

6. There is an important correlation between motivation for work and employability.

7. Cardiac anxiety is a factor in employability, but not a major one.

8. Therapeutic cardiac classification appears somewhat more indicative of employability than the functional classification. In our experience Class C patients, while employable, are less apt to be definitely so.

9. The number of prior infarcts alone is not an important determinant of employability.

10. A history of angina pectoris and the presence of hypertension appear to influence employability relatively little.

11. While marked cardiac enlargement

makes employment a bit less likely, heart size is not in itself an important factor

12. A history of congestive failure and the use of mercurial diuretics tend to decrease employability but are not bars to it

13. The degree or nature of electrocardiographic abnormalities is not of special significance in determining employability

14. The patients deemed employable, but who have not found employment, show factors to indicate that it is not the severity of the heart disease that is the cause, but rather factors of motivation and lack of work skill and of higher education

15. No clear-cut profile for employability of myocardial infarction cases can be established, especially on the basis of cardiac findings in themselves

16. We are left with the previously stated belief that the over-all picture of the whole patient, with major emphasis on a detailed medical history and examination, including observation of the patients' reactions to activity, tempered by clinical insight and experience, remains as our primary basis for determining employability.

SUMMARY AND GENERAL CONCLUSIONS

The emphasis of the study reported in this paper has been upon coronary heart disease with myocardial infarction. While not specifically demonstrated here, we are certain that

general factors regarding employability of all cardiacs are similar to those demonstrated for the coronary patient. Not infrequently we see two cardiacs highly comparable in age, nature of lesions, and apparent cardiac capacity, one may be employable and the other may not, one may be capable of undertaking a job of rather strenuous proportions while the other can do only sedentary work. All the variables of motivation, anxiety, experience, training, habits of years, and dexterity are the determinants.

It appears that some 60 per cent of cardiacs as seen by us may be employable, whether they have rheumatic, congenital, or degenerative heart disease. It is possible to find suitable employment for some 60 per cent of the *employables* with the assistance of proper counseling. By and large, evidence would indicate that they can tolerate work well without deleterious effect on their cardiovascular systems.

It is our opinion that patients may be satisfactorily evaluated for selective placement by ordinary clinical methods in use in most cardiac clinics, without elaborate or time-consuming functional studies. Unquestionably the application of the co-ordinated team approach is most highly desirable in reaching the desired end of employment. We believe that actual assistance toward employment should be carried through, preferably by members of the team as a sequel to evaluation.

CHAPTER 37 EXPERIENCE AT THE CARDIAC WORK CLASSIFICATION UNIT OF THE HEART ASSOCIATION OF SOUTHEASTERN PENNSYLVANIA (PHILADELPHIA)

David Gelfand

The Cardiac Work Classification Unit in Philadelphia, under the sponsorship of the Heart Association of Southeastern Pennsylvania and the Division of Adult Cardiovascular Diseases, Commonwealth of Pennsylvania, began in February, 1952. It was the decision of the organization committee that such a unit could be most productive and useful if admission policies were confined principally to persons who were employed or were considered to be employable. A rapport with industrial firms in and around Philadelphia was established for the referral of their problems of workers with heart disease. Patients were referred from 56 different industrial and business firms.

In order to avoid loss of time for employed referrals and since the Unit personnel were engaged primarily at their own work, the

weekly sessions were held at 6:30 P.M. It was felt that better service to the patient and to the referring source could be accomplished by completing the interviews, examinations, group conference, and recommendations in one session. This has been accomplished with only two exceptions in all cases. Admissions are limited to four new cases at one session. The Unit consists of a cardiologist, a psychiatrist, a medical social worker, and a vocational counselor. In 1953, a second Unit, composed of the same disciplines but not the same persons, was established to conduct only follow-up examinations.

It must be stressed that the multidiscipline approach was practiced and that no recommendation was made individually before the group conference was held. Strict adherence to the philosophy of the team approach en-

TABLE 37-1. AGE, SEX, RACE, SOURCE OF REFERRAL AND JOB STATUS ON INITIAL EVALUATION OF 616 CASES

Age group		Sex		Race		Source of referral				Job status on initial evaluation			
Age	Number of cases	Male	Female	White	Negro	Industry	Private doctor	Bureau of Rehabilitation	Other sources	Employed full time	Employed part time	Sick leave	Unemployed
20 & under	26 (4.1%)	17	9	24	2	20	4	0	2	16	0	2	8
21 to 34	63 (10.2%)	50	13	47	16	43	17	2	1	49	0	5	9
35 to 54	313 (51.0%)	276	37	276	37	223	69	11	10	181	6	82	44
55 & over	214 (34.7%)	203	11	195	19	160	41	3	10	119	9	62	24
Total	616	546 (88.8%)	70 (11.2%)	542	74	446 (72.4%)	131 (21.2%)	16 (2.6%)	23 (3.8%)	165 (26.8%)	15 (2.4%)	151 (24.5%)	85 (13.8%)

TABLE 37-2. RECOMMENDATIONS MADE TO 519 (84.2%) CARDIACS ON INITIAL EVALUATION

Age group		Unit recommendations on initial evaluation					
Age	Number of cases	Work without restrictions	Same work (restrictions)	Increased activity allowed	Reduce activity but full time return to work	Extend convalescence with or without care, e.g., surgery	Unable to work
20 and under	11 (2.1%)	3	5	0	1	1	1
21 to 34	39 (7.4%)	9	17	1	8	3	1
35 to 54	262 (50.5%)	28	131	10	69	18	6
55 and over	207 (40.0%)	9	122	6	55	7	8
Total	519	49 (9.5%)	275 (53.0%)	17 (3.3%)	133 (25.6%)	29 (5.6%)	16 (3.0%)

abled all members of the team to gain experience and insight into the problems of the worker with heart disease, the resulting disability and its relationship to employability*.

The composition, source of referral, and job status of 616 persons who were evaluated by the Unit between February, 1952, and December, 1956, are given in Table 37-1. Four hundred forty-six (72.4 per cent)

* We are particularly indebted to Miss Hope Thomson and Mr. John Hagan, who have been with the Unit since its inception, and to Dr. M. C. J. Pfeiffer, Dr. R. Monkett, Dr. J. Urbach, Dr. S. Bullock, Dr. T. M. McMillan, III, Dr. S. Shapiro, Dr. D. Lewis, Dr. J. Bryfogle, Dr. P. Ottenberg, Dr. W. S. Robbins, Miss R. Carp, Mrs. J. Wallace, Miss R. Brown, and Miss L. Harnick for their assistance in the operation of the Unit.

patients were referred to the Unit by industry (industrial physicians, personnel, and labor unions). This source of referral accounts for the fact that 531 (86.2 per cent) of the patients were working full or part time or were on sick leave from full-time jobs at the time of initial examination. The ratios of males to females, whites to nonwhites, and age distribution are comparable to other groups and no comment will be made.

Ninety-seven (18.8 per cent) patients were found to have no heart disease. This relatively low figure reflects good screening by the referring sources. The recommendations made to 519 cardiacs (Table 37-2) were that 474 (91.4 per cent) return to full time work,

TABLE 37-3 AGE, SEX, RACE, SOURCE OF REFERRAL, AND JOB STATUS OF 374 FOLLOW-UP CASES

Age group		Sex		Race		Source of referral				Job status on initial evaluation			
Age	Number of cases	Male	Female	White	Negro	Industry	Private doctor	Bureau of Rehabilitation	Other sources	Employed full time	Employed part time	Sick leave	Unemployed
20 & under	10 (2.5%)	7	3	8	2	7	2	0	1	4	0	1	5
21 to 34	44 (11.9%)	35	9	35	9	32	11	1	0	36	0	3	5
35 to 54	193 (51.6%)	170	23	173	20	136	44	6	7	104	6	56	27
55 & over	127 (34%)	119	8	119	8	92	27	1	7	76	4	35	12
Total	374	331	43	335 (88.5%)	39 (11.5%)	267 (71.5%)	84 (22.5%)	8 (1.9%)	15 (4.1%)	220* (58.8%)	10† (2.7%)	95 (25.4%)	41‡ (10.9%)

* Six applying for new jobs

† One applying for a new job

‡ Twelve applying for jobs

TABLE 37-4 RECOMMENDATIONS MADE TO 331 CARDIACS ON INITIAL EVALUATION

Age group		Unit recommendations on initial evaluation					
Age	Number of cases	Work without restrictions	Same work (restrictions)	Increased activity allowed	Reduce activity but full time return to work	Extend convalescence with or without care, e.g., surgery	Unable to work
20 and under, age group I	6 (1.9%)	2	1	0	1	1	1
21 to 34, age group II	31 (9.4%)	8	13	1	6	2	1
35 to 54, age group III	171 (51.7%)	18	85	4	48	12	4
55 & over, age group IV	123 (37.0%)	5	75	4	30	5	4
Total	331	33 (10.0%)	174 (52.6%)	9 (2.7%)	85 (25.7%)	20 (6.0%)	10 (3.0%)

with or without restrictions; in some instances the job could be modified to conform to the recommended restrictions, in others a change of job was accomplished.

Table 37-3 represents the composition, source of referral, and job status (on initial evaluation) of a group of 374 cases who had one or more follow-up examinations. Comparison with Table 37-1 shows little or no change in the composition of these groups in any category, including ages, sex, race, source of referral, and employment status.

The Unit recommendation (Table 37-4) made to 331 patients with heart disease who

had one or more follow-up examinations was that 301 (91 per cent) could return to full time work. Note again that there is no change statistically in comparison with Table 37-2.

The employment status on initial and follow-up evaluation in 374 patients is given in Table 37-5. There was an increase in 56 (14.9 per cent) patients who were employed despite the retirement of 23 patients (22 of whom were employed or on sick leave on initial examination). The changes were in the cases on sick leave and unemployed when first evaluated. Of the 95 cases on sick leave,

TABLE 37-5 EMPLOYMENT STATUS ON INITIAL AND FOLLOW-UP EVALUATION OF 374 CASES

<i>Initial work status</i>			<i>Follow-up status</i>			
	<i>Status</i>	<i>Number of cases</i>	<i>Employed</i>	<i>Sick leave</i>	<i>Unemployed</i>	<i>Retired</i>
Cardiacs 331 (88.5%)	Employed	198 (53.0%)	168	6	14	10
	Sick leave	90 (24.0%)	52	12	14	12
	Unemployed	43 (11.4%)	25	0	17	1
No heart disease 43 (11.5%)	Employed	32 (8.6%)	32	0	0	0
	Sick leave	5 (1.4%)	5	0	0	0
	Unemployed	6 (1.6%)	4	0	2	0
Total		374	286 (76.5%)	18 (5.0%)	47*†‡ (12.4%)	23 (6.1%)

* Housewives (7).

† Psychopathic inferior

‡ Applying (4).

TABLE 37-6 OCCUPATION ON INITIAL AND ON FOLLOW-UP EVALUATION OF 251 CARDIACS WHO WERE WORKING AT TIME OF FOLLOW-UP

<i>Occupation on initial evaluation</i>		<i>Occupation on follow-up evaluation</i>						
<i>Occupation</i>	<i>Number of cases</i>	<i>Professional managerial</i>	<i>Clerical sales</i>	<i>Service</i>	<i>Skilled</i>	<i>Semi-skilled</i>	<i>Unskilled</i>	<i>Housewives</i>
Professional managerial	22	21			1			
	(9.0%)							
Clerical sales	16	1	32	2		1		
	(14.3%)							
Service	28			27		1		
	(10.5%)							
Skilled	69	3	4	4	53	5		
	(27.5%)							
Semi-skilled	54		4		2	40	6	2
	(22.0%)							
Unskilled	37		1	3	1	6	25	1
	(14.7%)							
Housewives	4			1				3
	(1.6%)							
Never worked	1						1	
	(0.4%)							
Total	251	25	41	37	57	53	32	6
	(100%)							

57 (60 per cent) returned to work, of the 49 unemployed patients, 29 (60 per cent) were able to get full-time work. Thirty-five of those initially on sick leave were in the age group of 55 years and over, of these, 17 re-

turned to work. Of the 23 who retired, 18 were in the age group of 55 years or over.

Table 37-6 compares the occupations on initial evaluation with the occupations on follow-up evaluation of the 251 cardiac cases

TABLE 37-7 MEDICAL STATUS ON FOLLOW-UP EVALUATION RELATED TO EMPLOYMENT STATUS ON INITIAL AND FOLLOW-UP EVALUATION OF 331 CARDIAC CASES

Initial work status		Follow-up work status		Medical status		
Status	Number of cases	Status	Number of cases	Improved	Same	Worse
Employed	198	Employed	168	32*	113	23†
		Sick leave	6	1	4	1
		Unemployed	14	2	7	5
		Retired	10	1	3	6
Sick leave	90	Employed	52	17†	31	4
		Sick leave	12	3	6	3
		Unemployed	14	3	10	1
		Retired	12	2	6	4
Unemployed	43	Employed	25	9†	12	4
		Sick leave	0	0	0	0
		Unemployed	17	6†	9	2
		Retired	1	1	0	0
Total	331		331	77	201	53

* 4 cases had commissurotomies

† 1 case had commissurotomy

TABLE 37-8 MEDICAL STATUS AND EMPLOYMENT STATUS ON FOLLOW-UP EVALUATION RELATED TO DIAGNOSIS OF 331 CARDIAC CASES

Diagnosis		Medical status on follow-up					
		Improved		Same		Worse	
Heart disease	Number of cases	Working	Not working	Working	Not working	Working	Not working
Congenital	4 (1.0%)	0	0	4	0	0	0
Rheumatic	70 (21.0%)	17	4	22	9	10	8
Hypertensive	64 (20.0%)	9	3	33	9	9	1
Arteriosclerotic	39 (12.0%)	0	1	19	8	3	8
Coronary artery	142 (43.0%)	29	11	65	26	7	4
Other	12 (3.0%)	3	0	6	0	2	1
Total	331	58 (16%)	19 (7%)	149 (39%)	52 (21%)	31 (10%)	22 (7%)

who were found to be working at the time of follow-up examination. That the employer need not lose the skill and experience of an employee who develops organic heart disease is supported by the fact that a high percentage of the patients remained in the same job category following evaluation by the Unit. A few cases were placed in jobs where the

physical activity was less but with greater responsibility. The greatest problems were encountered in the unskilled occupations that required great physical exertion that might aggravate the cardiac condition of the patient. In these cases the lack of job opportunities and the lack of skill and of training compounded the problems encountered.

The question of how well the patient does clinically when working and when not working is summarized in Table 37-7. Of the 331 patients with heart disease who had a follow-up examination, the number employed increased from 198 on initial examination to 255 on follow-up examination. This was the result of 52 patients returning to work from sick leave and 25 patients obtaining employment after evaluation by the Unit. The fact that work does not necessarily aggravate heart disease is demonstrated in the second part of Table 37-7. Of the 77 patients found to be physically improved on follow-up eval-

heart disease, 49 per cent of the patients with arteriosclerotic heart disease, and 66 per cent of the patients with coronary artery disease were improved or the same medically while working. Eighteen (34 per cent) of the 53 patients found to be medically worse on follow-up examination had a diagnosis of rheumatic heart disease. Of these, 6 patients were in age Group II (21-34 years) and 10 patients were in age Group III (35-54 years). This discouraging prognosis, in a relatively young and productive age group, may be reversed by early diagnosis and more definitive treatment (e.g., cardiac surgery).

TABLE 37-9 DIAGNOSIS AND REASON UNEMPLOYED OF 69 UNEMPLOYED CARDIACS

Heart disease	Diagnosis Number of cases	Reason unemployed					
		Unable to get job	Unwilling to take job available	Unable to hold job	Temporary cardiac illness	Lay-off	Retired
Congenital	0						
Rheumatic	22 32.0%	4	13		4		1
Hypertensive	4 (5.0%)	1				1	2
Arteriosclerotic	12 (15.0%)		1	3			8
Coronary artery	30 (46.6%)	9	7		3	1	10
Other	1 (1.4%)			1			
Total	69	14	21*	4	7	2	21

* Seven housewives.

uation, 58 (78 per cent) were working, of the 201 patients found to be in the same physical condition, 156 (77 per cent) were working. In the 53 cases who were found worse, 31 (60 per cent) of whom were working, it was felt that the natural progression of the heart disease was responsible, rather than the physical exertion of the job.

The relationship of the diagnosis to the medical status on follow-up examination, comparing those patients who were working with those not working, is given in Table 37-8. It is of interest to note that 56 per cent of the patients with rheumatic heart disease, 65 per cent of the patients with hypertensive

especially with the development and improvement of such surgical techniques.

An analysis of 69 patients with disease (Table 37-9) who were unemployed showed that 32 per cent of the rheumatic heart disease (17 of 53 patients were in age Groups I, II, and 46.6 per cent had coronary (15 of the 30 patients were over age). Hypertensive heart disease presents a relatively small proportionally since only 4 patients of this group had this condition.

Although it is the patients'

cases are closed from time to time. Table 37-10 shows the reason for closing 112 (18 per cent) of the total of 616 cases evaluated from February, 1952, through December, 1956. Unfortunately, 45 patients did not have a

means of a questionnaire and, in most cases, cover the period of time from initial evaluation by the Unit to the date of the last follow-up evaluation. The patients with heart disease lost 8.8 days per 100 scheduled work-

TABLE 37-10. REASON FOR CLOSING 112 CASES
(18% of 616 Cases)

<i>Follow-up status</i>		<i>Reason for closing</i>					
<i>Follow-up</i>	<i>Number of cases</i>	<i>Deceased</i>	<i>Retired</i>	<i>No heart disease</i>	<i>Will not return</i>	<i>Address unknown</i>	<i>Other reasons*</i>
Had one or more follow-up evaluations	67 (60%)	23	7	35	1		1
Had no follow-up evaluation	45 (40%)	28	5	1	3	6	2
Total	112 (100%)	51 (45.5%)	12 (10.7%)	36 (32.0%)	4 (3.6%)	6 (5.4%)	3 (2.8%)

* Other diseases such as cancer, unemployment because of mental incapacity.

TABLE 37-11 ABSENTEEISM ON 191 CASES

<i>Diagnosis</i>	<i>Number of cases</i>	<i>Work weeks</i>	<i>Days lost because of</i>			
			<i>Cardiac illness</i>	<i>Injury on job</i>	<i>Other illness</i>	<i>Personal reasons other than illness</i>
Congenital and other heart disease	13	933	34	0	33	10
Rheumatic	26	1,677	438	43	101	43
			Commis., 117 days			
Hypertensive	43	3,185	666	78	364	202
Arteriosclerotic	22	1,247	521	0	227	70
Coronary artery	67	3,398	1,251	38	370	114
Total cardiac	171	10,440 weeks or 52,200 work days*	2,910	159	1,025	439
No heart disease	20	1,533 weeks or 7,665 work days*		0	291	86
Total	191	11,973 or 59,865 work days	2,910		1,386	525

* Five days per week

follow-up evaluation prior to closing. The largest number of cases were closed because of death and the next largest number because of the finding of no heart disease.

Absenteeism figures (Table 37-11) are available on 171 patients with heart disease and 20 patients without heart disease. These figures were obtained from the employer by

ing days: 5.4 days for cardiac illness, 0.4 days for injury sustained on the job, 2.1 days for other illness, and 0.9 days for personal reasons. The 20 noncardiac patients lost 4.9 days per 100 scheduled working days: 3.8 days for illness and 1.1 days for personal reasons. No absenteeism figures are available on the working population of the Philadelphia area,

but the information given above may be compared with a national figure of 3.4 days lost by unimpaired workers per 100 scheduled working days, given in Bulletin 923 of the Bureau of Labor Statistics of the United States Department of Labor.

SUMMARY

Our Moderator has requested "facts, not presumptions; and numbers, not words." These have been given. However, it must be

pointed out that in slightly over 46 per cent of the patients seen, physical factors and job opportunities were less important than social, economic, cultural, and emotional factors in employment motivation and vocational adjustment. This Unit, by the full use of the multidiscipline approach to rehabilitation, has been able to recognize and in many cases to help adjust some of the factors that have prevented the maximum utilization of the patients' vocational capabilities.

CHAPTER 38 RESULTS OF STUDIES AT THE WORK CLASSIFICATION CLINIC OF THE CLEVELAND AREA HEART SOCIETY

T. V. Parran, H. K. Hellerstein, D. Cohen, and E. Goldston

This report presents a statistical analysis of some of the experience of The Work Classification Clinic of The Cleveland Area Heart Society in evaluating persons with real or suspected heart disease, and in assisting them to return to competitive, productive occupations.

The philosophy, composition, purposes, and modus operandi of The Work Classification Clinic in Cleveland have been presented by one of us (1) and are basically similar to those originated by Dr. Leonard Goldwater in 1941, at Bellevue Hospital in New York.

MATERIALS AND METHODS

The records of 595 cases were selected at random from the initial 1,000 patients referred during the first six years of the Clinic's operation. A total of 1,272 new patients have

been studied to date. During this interval, 3,526 follow-up examinations and 4,798 patient visits were made. A coding system has been used since the Clinic was founded to provide detailed information (78 items with

TABLE 38-1 DISTRIBUTION OF CASES OF THE CLEVELAND WORK CLASSIFICATION CLINIC

(June to June)	Number of new cases	Number of cases in present study	Per cent analyzed
1950-1951	155	117	75.5
1951-1952	123	103	84.1
1952-1953	132	85	64.4
1953-1954	213	105	49.3
1954-1955	225	102	45.3
1955-1956	232	83	35.8
May 1956-1957	192	0	0
Total	1,272	595	

provision for 9 variables), giving a comprehensive picture of the medical, social, and vocational aspects of the study and of the follow-up examination. This information was transferred to IBM code cards for statistical analysis. The distribution of the 595 cases analyzed is shown in Table 38-1. The present report consists of the data of the initial evaluation and the most recent follow-up examination of these 595 cases.

SOURCE OF REFERRAL

Private physicians referred 66.4 per cent of the patients, industrial physicians 17.8 per cent, hospital and clinic physicians 9.8 per cent, social agencies 4.9 per cent, and 1.2 per cent of the referrals were self-initiated. Joint referrals submitted by both the industrial and private physicians were considered initiated by the industrial physician. Several large industries in the Cleveland area have recently adopted the policy of sending all employees to the Work Classification Clinic after a heart attack for evaluation before returning them to work. Referring companies represent a cross section of Cleveland industry, manufacturing, transportation, public service, government, wholesale and retail trade, and construction.

DESCRIPTION OF PATIENTS

From the medical, social, and vocational standpoints, the patient load is considered to be representative of people with heart disease in Cleveland.

Age, Sex, and Color

The preponderance of males in this study (88.0 per cent males, 12.0 per cent females) is consistent with that in industry and in the general population with heart disease. The color distribution was 90.4 per cent white and 9.6 per cent Negro (In Cleveland, 23 per cent of the general population is Negro). The population was a mature, adult population, with 65.5 per cent of the group 45 years and older (Table 38-2). The group with degenerative heart disease was the oldest. Sixty-two per cent of the rheumatic heart

TABLE 38-2 AGE AND ETIOLOGIC CLASSIFICATION

Etiology	20	20	35	40	45	50	55	60	65	Total
	yrs	to 34	to 44	to 49	to 54	to 59	to 64	to 69	or over	number
Rheumatic	8	30	18	12	19	13	6	2	1	107
Congenital	5	8			1					14
HCVD*		2	2	50	11	50	4	7	1	147
HCVD and ASHD†										
ASHD		1	1	19	13	19	27	29	9	118
Cor pulmonale		4	11	28	49	42	44	34	19	231
Syphilitic		1	3	1		2	4	1	2	14
Etiology unknown				1	2	3		2	1	9
No heart disease	2	9	5	1	4	1	1			21
		15	3	5	3	2	1		1	32
Total	15	70	43	77	102	92	87	75	34	575
Per cent	2.5	12.2	7.5	13.4	17.7	16.1	15.1	13.1	6.0	
Number		205			281			503		
Per cent		34.5			47.2			18.3		

* HCVD, Hypertensive cardiovascular disease.

† ASHD, Arteriosclerotic heart disease.

disease group, 19 per cent of the degenerative heart disease group, and 79 per cent of the patients with no heart disease were younger than 45 years of age.

Etiologic Classification

The etiologic types are representative of those usually encountered in clinical practice. The most common was degenerative heart disease with 18.5 per cent in the degenerative group, 66.8 per cent in the degenerative group, (39 per cent with arteriosclerotic heart disease, 19.9 per cent with hypertensive and arteriosclerotic heart disease, 2.4 per cent with hypertensive (arteriosclerotic disease), 2.4 per cent with congenital cardiac anomalies, 2.4 per cent with syphilitic heart disease, 2.4 per cent with no heart disease, and 2.4 per cent with heart disease of undetermined type). Twenty-eight cases were multiple and were classified under the primary diagnosis.

Functional and Therapeutic Classification

When classified according to the New York Heart Association classification, 10 per cent of the patients were placed in Class I, 19 per cent in Class II, 19.5 per cent in Class III, and 4.7 per cent in Class IV. One patient with no heart disease was classified III C on a noncardiac basis. The proportion of patients in each class is shown in Table 38-3.

than in the degenerative group were classified as III C or worse (Table 38-3). The relationship of functional and therapeutic classification to work status and follow-up mortality will be discussed later.

Medical treatment was estimated to be adequate in 78 per cent and inadequate in 20 per cent of the patients at the time of the initial visits. The adequacy of the medical

cases and especially common in the group without heart disease (62.5 per cent). Obesity occurred in 19.5 per cent of the entire series in 4.5 per cent of the rheumatic group, and 26 per cent of the degenerative group. Gall bladder disease and peptic ulcer, present or past, was noted in 16 per cent of all cases occurring in 24.2 per cent of those with arteriosclerotic heart disease, 17.0 per cent of

TABLE 38-3 FUNCTIONAL AND THERAPEUTIC CLASSIFICATION

Etiology	Classifications				Possible and potential	Not classified	Total
	I A,B	I C	II B,C	III C			
Rheumatic	20	2	48	28	10	1	109
Congenital	9		5				14
HCVD	13	2	23	6	2	1	47
HCVD and ASHD	9	5	68	28	7	1	118
ASHD	48	7	127	40	6	3	231
Cor pulmonale			6	7	1		14
Syphilitic	2		3	3	1		9
Etiology unknown	15		1	4	1		21
No heart disease	24			1		5	32
Total	140	16	281	117	28	8	595
Per cent	23.5	2.7	47.5	19.5	4.7	1.3	100.0

care did not vary significantly from this ratio for the various etiologic groups.

Family History of Heart Disease

A reliable detailed family history was obtained in 504 subjects. Similar heart disease was present in 56.4 per cent of the hypertensives, in 51.5 per cent of those with arteriosclerotic heart disease, in 70.5 per cent of the subjects with both arteriosclerotic and hypertensive cardiovascular disease, and 23.7 per cent of those in the rheumatic group. Ten of the 28 cases in the group with no heart disease gave a reliable family history of heart disease. Similar heart disease was present in the family histories of 3 cases in the congenital group.

Associated Diseases of Significance

The most common and probably the most important associated disease state was that of emotional difficulty (anxiety, tension, alcoholism, etc.), occurring in 39 per cent of the

the hypertensives with arteriosclerotic heart disease, and 8.2 per cent of the patients with rheumatic heart disease. Significant pulmonary disease was present in 12 per cent of the cases. Musculoskeletal disability, arthritis, peri-arthritis, or post-infarction shoulder-hand syndrome was noted in 9 per cent, diabetes mellitus in 5 per cent, and renal disease in 6 per cent of the cases.

Somatotypes

On each patient's initial visit a rough estimate of body types was made by the cardiologists, and the patients were classified using the method of Sheldon, Dupertuis, and McDermott.² Correlations between this rough estimate and more precise determinations performed by Dr C. W. Dupertuis have proved to be surprisingly good. The degenerative group showed predominant endomorphism (50 per cent), while the subjects with rheumatic mitral disease were predominantly ectomorphomorphic (Table 38-4).

TABLE 38-4 BODY TYPE AND HEART DISEASE

Diagnosis	Endomorph	Ectomorph	Mesomorph	Ectomorph	Ectomorph	Vol done	Total
Rheumatic	3	17	18	50	18	3	109
Congenital		2	3	5	4		14
HCVD	3	22	9	11	1	1	47
HCVD and ASHD	6	67	20	23		2	116
ASHD	5	107	44	68	7		231
Cor pulmonale		4		7	2	1	14
Syphilitic		5	1	2		1	9
Etiology unknown	1	3	6	9	1	1	21
No heart disease	1	9	7	10	4	1	32

Exercise Tolerance Test (Table 38-5)

Exercise tolerance tests were considered valuable in providing some objective information about the patient's estimated work capacity. The standard Master exercise tolerance test over two 9-inch steps exacts an expenditure of energy of 8 calories per minute, which is a significant peak load, rarely exceeded in light or moderate occupations.¹

Response to exercise was considered abnormal if symptoms or signs of cardiopulmonary embarrassment appeared. These included (1) pain, angina, marked dyspnea, cyanosis or venous distention, (2) failure of the cardiac mechanism, sinus tachycardia with a heart rate above 125 per minute, paroxysmal tachycardia or arrhythmia or premature beats, (3) electrocardiographic evidence of unpaired conduction, i.e., A-V block or bundle branch block, (4) coronary insufficiency as indicated by ST-T changes in the electrocardiograms (in absence of digitalis medication), and (5) persistence of tachycardia after 3 minutes of rest (Table 38-5).

Exercise tests were performed routinely in all cases except in the presence of uncompensated congestive failure, recent myocardial infarction (within 3 months), or possible impending infarction (recently increasing angina pectoris). The Master exercise test was performed in 453 of 595 cases. The response was abnormal in 34.2 per cent and normal in 65.8 per cent. Abnormal responses were obtained in all categories with heart disease. The nonspecificity of the test for coronary disease is shown by the fact that a similar

number of the rheumatic and arteriosclerotic patients had abnormal responses (Table 38-5). Extensive myocardial infarction was not necessarily associated with a poor tolerance to the prescribed exercise of the test. Pa-

TABLE 38-5 EXERCISE TOLERANCE TESTS

Etiologic classification	Number tested	Response	
		Abnormal	Normal
Rheumatic (109)	76	34.2	65.8
Congenital (14)	10	20.0	80.0
HCVD (47)	36	13.9	86.1
HCVD and ASHD (118)	85	41.2	58.8
ASHD (231)	190	40.0	60.0
Cor pulmonale (14)	7	57.0	43.0
Syphilitic (9)	8	50.0	50.0
Unknown etiology	17	17.6	82.4
No heart disease (32)	24	0	100.0
Total (595)	453	34.2 (155 cases)	65.8 (298 cases)

tients with large ventricular aneurysms have had a normal response to this exercise test.

Vocational Data at Initial Visit

At the time of the initial visit, 40 per cent of the patients were employed, 60 per cent unemployed. This distribution was similar in

the various etiologic groups, except in the cor pulmonale group, where 12 of the 14 cases were unemployed (Table 38-6).

TABLE 38-6 EMPLOYMENT STATUS BY ETIOLOGIC CLASSIFICATION, FIRST VISIT TO CLEVELAND WORK CLASSIFICATION CLINIC

Etiology	Em- ployed	Unem- ployed	Un- known	Number
Rheumatic	36	71	2	109
Congenital	8	6		14
HCVD	12	35		47
HCVD and ASHD	44	74		118
ASHD	103	128		231
Cor pulmonale	2	12		14
Syphilitic	4	5		9
Undiagnosed	11	9	1	21
No heart disease	18	13	1	32
Total	238	353	4	595
Per cent	40.0	59.3	0.7	100.0

Employment status correlated closely with functional and therapeutic classification (Table 38-7). Only 30 per cent of the cases with I A classifications were unemployed, while 86 per cent with a classification of worse than III C were unemployed, with a progressive increase in unemployment percentage from the I A to the worse than III C classification. It is interesting that 4 of 5 cases with a classification of possible or potential heart disease were unemployed.

VOCATIONAL CHARACTERISTICS The employment history obtained at the initial visit indicated a stable work background. Fifty-six

per cent of the patients had worked 5 years or more at the last place of employment, 39 per cent more than 10 years, and 23 per cent more than 20 years.

The educational background of the patients varied. Twenty-three per cent of the patients had completed less than 8 years of schooling, 29 per cent had completed high school, 8.7 per cent had completed at least 2 years of college, and 1.7 per cent had attained postgraduate status.

The variety of the occupational and industrial classifications of the patients reflects the breadth of the clinic experience and indicates that heart disease occurs in all occupations (Table 38-8). All levels of occupational classifications with the exception of farming were represented, from the top level managerial and professional to the unskilled laboring groups. Of the jobs held by patients in this series, 397 could be categorized as to skill. Thirty-seven per cent of these were classified as skilled, 43 per cent semiskilled, and 20 per cent unskilled.

As in the more common occupa-

tions than did any other etiologic group. All industrial classifications were represented in the patient load, but four times as many patients were listed in the *All Manufacturing* category as in any other group (Table 38-9).

A significant change in occupational or industrial classification as a result of illness was

TABLE 38-7 RELATIONSHIP OF EMPLOYMENT STATUS TO FUNCTIONAL AND THERAPEUTIC CLASSIFICATION AT INITIAL VISIT

Classification	Number	Employed, number	Per cent	Unemployed, number	Per cent
I A	49	34	70	15	30
I B	90	53	59		41
I C	15	7	47	8	53
II B	148	60	40	88	60
II C	130	47	36	83	64
III C	116	31	27	85	73
Worse than III C	28	4	14	24	86
Possible and potential	5	1	20	4	80
Unclassifiable	6	1	17	5	83
Not classified	8			4	50

TABLE 38-8. OCCUPATIONAL CLASSIFICATION BEFORE ILLNESS

<i>Occupational classification</i>	<i>Number</i>	<i>Per cent</i>
Professional occupation	22	4
Semiprofessional	11	2
Managerial & official	31	5
Clerical & kindred	54	9
Sales & kindred	53	9
Domestic service	11	2
Personal service	29	5
Protective service	24	3
Service work (porter, etc.)	23	3
Agriculture, horticulture	1	
Skilled	147	25
Semiskilled	172	29
Unskilled	78	13
Housewife	14	2
Unemployed	12	2
Self-employed	36	6
Not classified	26	4
Total	744	

TABLE 38-9. INDUSTRIAL CLASSIFICATION BEFORE ILLNESS

<i>Industrial classification</i>	<i>Number of cases</i>	<i>Per cent</i>
Farming and mining	4	0.7
Contract and construction	25	4.0
All manufacturing	281	47.0
Transportation and communication	43	7.0
Wholesale, retail	75	13.0
Finance, real estate, and insurance	12	2.0
Service industry	51	8.5
Government	31	5.0
Not elsewhere classified	41	7.0
Not classified	32	5.5
Total	595	

observed in slightly more than half of the patients, creating a need for careful vocational evaluation.

Social Factors

From a social standpoint, the population was representative of urban Cleveland, being medically nonindigent (96.0 per cent), married with dependents (89.0 per cent), and beset by financial responsibilities and pressures in approximately 65.0 per cent, despite pension plans and unemployment insurance.

The patient's attitude toward the clinic at the initial visit varied, depending upon the possible significance of the clinic's findings. The attitude was friendly, however, by a seven to one ratio. Hostility, when present, appeared to result from the notion that the clinic represented either a threat to continued employment or pressure toward employment, depending upon the employment status of the patients who exhibited this attitude.

Anxiety was strikingly important in all categories. Forty to 50 per cent of the patients in all etiologic groups were apprehensive beyond the reality of the situation. The genesis of this anxiety has not been analyzed statistically in the present study. The capacity to accept the impact and the fact of heart disease was apparently unrelated to the employment status. Approximately 60 per cent of both employed and unemployed subjects were unable to accept their illness intellectually and emotionally. Twenty-eight per cent of the patients were excessively pessimistic about job opportunities. Only 4 per cent were considered to be hypomotivated. The arteriosclerotic heart disease group was particularly hypermotivated.

DISPOSITION OF CASES AFTER INITIAL VISIT

The disposition of each case was much more complex than merely making recommendations, and was dependent mainly upon the needs determined at the initial visit. Reassurance on a supportive level was indicated in 71.0 per cent of the cases. Fifty-eight per cent were felt to need intellectual interpretation of the medical situation. Vocational guidance, with varying degree of thoroughness, was required by 42 per cent, vocational rehabilitation by 12 per cent, and physical rehabilitation to improve work tolerance in 7 per cent. Need for emotional rehabilitation was recognized in 28 per cent of the cases.

The needs were many in all etiologic categories, and usually multiple in individual cases (Table 38-10). The rheumatic group

TABLE 38-10 APPARENT NEEDS AT INITIAL EVALUATION

	<i>RHD</i> (107)	<i>Congenital</i> (14)	<i>Degenerative</i> (306)	<i>Cor</i> <i>pulmonale</i> (74)	<i>Syphilis</i> (9)	<i>Unknown</i> <i>H D</i> (27)	<i>V</i> <i>H D</i> (35)
	%	%	%	%	%	%	%
Vocational guidance	48	57	41	50	45	33	19
Vocational rehabilitation	23	14	8	45	11	10	0
Physical rehabilitation	10	7	6	14	0	5	0
Avocational guidance	1	0	2	20	0	5	0
Emotional rehabilitation	31	14	28	21	0	23	44
Intellectual interpretation	56	43	60	43	67	71	59
Reassurance	65	50	73	79	55	62	78
More medical RX	32	7	15	14	45	24	6
More diagnosis	3	14	3	0	0	14	9

specifically showed a greater need for more extensive vocational and physical rehabilitation and more intensive medical treatment than did any of the other etiologic groups, owing to the younger age, shorter and more restricted vocational experience, and associate embolic neuromuscular complications.

Vocational recommendations were likewise many and multiple in individual cases. The present occupation or employment status was judged satisfactory in 35.0 per cent of the cases; in each etiologic category, approximately equal numbers were graded up and down, i.e., 22 per cent and 27 per cent respectively. In 6.5 per cent of the total study, no employment was considered feasible. In the rheumatic group with a more limited functional capacity, this judgment was made in 11.9 per cent. The various recommendations were implemented by the appropriate member of the clinic team. Attempts were made to effect proper job placement by reassignment within the same plant or job modification when indicated. Job transfer was suggested in 18 per cent; referral to job placement services in 18 per cent; vocational counseling in 15.5 per cent; referral to sheltered workshop in 6 per cent; suggested formal psychotherapy in 7 per cent, and referral to family or other social agency in 2.5 per cent. Individualized interpretation of the medical situation and supportive reassurance were given to virtually all subjects. Subjects were referred to appropriate community facilities (Table 38-11). Seventeen per cent

TABLE 38-11. REFERRAL TO COMMUNITY AGENCIES AFTER INITIAL VISIT

<i>Agencies</i>	<i>Number</i>	<i>Per</i> <i>of cases</i>	<i>and</i>
Employment OSES, JVS, VGB	102	17.1	
Welfare CWB, DR, S&SR	11	1.9	
Family Service JFSA, FS	13	2.2	
Vocational service and training	59	10.0	
Physical rehabilitation CRC, BVR	49	8.3	
Hospitals	27	4.5	
Other	15	2.5	

of the cases were referred to employment agencies after the initial visit, and only 2 per cent were referred to welfare agencies.

FOLLOW-UP OBSERVATIONS

Follow-up study has been successful in all but 5 cases. Ten cases were discharged after the initial visit because there was no heart

TABLE 38-12 DURATION OF FOLLOW-UP

	<i>Number</i>	<i>Per cent</i>
1-6 months	94	15.8
6-12 months	88	14.8
1-2 years	176	29.6
2-3 years	117	19.7
3-4 years	64	10.7
4-5 years	15	2.5
5-6 years	26	4.4
No need for follow-up	10	1.7
Contact lost	5	0.8
Total	595	100.0

disease, and no reason existed for their follow-up. Of the remaining 585 patients, contact was lost in 5 cases. Thus, follow-up was obtained in over 98 per cent of the cases.

The duration of the follow-up is summarized in Table 38-12. In 30 per cent of the cases 12 months or less elapsed between the initial and last visit, 30 per cent 1 to 2

none of those with no heart disease. The functional and therapeutic classifications at the original clinic visit showed a prognostic trend, since on follow-up the mortality progressed with the classifications (Table 38-13). Groups I A and B had the lowest mortality, 8 per cent, one third of which was noncardiac, Groups I C and II B and C had a com-

TABLE 38-13 RELATIONSHIP OF FUNCTIONAL AND THERAPEUTIC CLASSIFICATION AT ORIGINAL CLINIC VISIT TO SUBSEQUENT MORTALITY

Classification on original visit	Original population	Died		Cause of death	
		Number	Per cent	Cardiac	Noncardiac
I A, B	140	11	8	7	4
I C	16	3	19	3	0
II B, C	281	47	17	46	1
III C	117	34	29	34	0
Worse than III C	28	13	46	12	1
Possible and potential	5	0	0	0	0
Nonclassifiable	8	3	38	3	0
Total	595	111	18.5	105	6

TABLE 38-14 CHANGE IN CARDIAC STATUS ON FOLLOW-UP

Etiology	Total	Died	Unchanged	Improved	Worse	Not classified	Deteriorated D & W	Per cent D & W
Rheumatic	109	25	45	25	14	3	39	35
Congenital	14	0	9	5	0	0	0	0
HCVD	47	8	24	8	7	0	15	32
HCVD and ASHD	118	31	43	18	31	2	62	59
ASHD	231	40	113	51	27	3	67	29
Cor pulmonale	14	3	5	4	2	0	5	36
Syphilitic	9	3	2	2	2	0	5	55
Undiagnosed heart disease	21	1	12	6	2	0	3	14
No heart disease	32	0	28	1	0	3	0	0
Total	595	111	281	120	83	11	196	32

years, 30 per cent 2 to 3 years, and 8 per cent 4 years or more. The follow-up duration for each etiologic group was comparable.

Mortality

Of the total study group of 595 cases, 111 cases (18.5 per cent) are known to have died, 23 per cent of the rheumatic group, 20 per cent of the degenerative group, 21 per cent of the cor pulmonale group, 5 per cent of those with undiagnosed heart disease, and

comparable mortality, 19 and 17 per cent respectively. Group III C, 29 per cent, and worse than III C, 46 per cent. Deaths were due to cardiac reasons in 105 cases, and noncardiac reasons in only 6 cases. Although no patient died on the job, 56 of the 111 died while employed.

Change in Cardiac Status on Follow-Up

In 65 per cent of the cases, the cardiac status was unchanged or improved (Table

38-14). Twenty per cent improved, 46 per cent remained unchanged, 14 per cent deteriorated, and 18.5 per cent died. If the last two groups are considered together, deterioration occurred in 32.5 per cent of the cases. This percentage is relatively constant for the various etiologic groups, with possible exception of the group with both hypertension and arteriosclerotic heart disease, where 50 per cent deteriorated. In contrast, the arteriosclerotic heart group deteriorated in 29 per cent, with a 17 per cent mortality. The relatively higher mortality rate of the rheumatic group is related to the initially poor classification.

Change in Occupational and Work Status on Follow-Up

The work status in the follow-up visit remained unchanged from that of the initial visit in 44 per cent, improved in 33 per cent, and deteriorated in 9 per cent. Thus, in the survivors (excluding those who died), the work status improved approximately 3.2 times as often as it deteriorated (Table 38-15). The improvement in work capacity was

TABLE 38-15 CHANGES IN CARDIAC AND EMPLOYED STATUS AT FOLLOW-UP AT CLEVELAND WORK CLASSIFICATION CLINIC

Status	Employment status (per cent)	Cardiac status (per cent)
Unchanged	44	47
Better	33	20
Worse	5	18
Died	18	18

estimated to be due to reassurance, interpretation, emotional rehabilitation, and improvement of cardiac function, occurring spontaneously after medical therapy or with physical rehabilitation in sheltered work shops.

The change in the employment status may be considered from the standpoint of the employment status at the time of the initial evaluation. The mortality rate of the unemployed group was higher than that of the employed group, i.e., 21.8 and 14.3 per cent respectively; as mentioned previously, the

heart disease was more advanced in the former group. In 64 per cent of the employed group, the employment status remained unchanged. In the unemployed group, 24.4 per cent remained unemployed, and 41.6 per cent obtained employment. At the follow-up visit, 70.5 per cent of the living subjects were employed.

Compensation or Death Claims

To date, there have been no known legal claims that illness or death was produced by employment of any of the subjects after evaluation by the Cleveland Work Classification Clinic.

SUMMARY

A statistical analysis has been made of data obtained on 595 subjects evaluated at the Work Classification Clinic of the Cleveland Area Heart Society. A team approach integrating medical, vocational, and social sciences has been used. The subjects were representative of a large cross section of Cleveland industry. They were adults, predominantly white and male, nonindigent, and had a stable yet varied work background. The etiologic categories of heart disease also were representative of clinical heart disease. Medical therapy and evaluation were considered adequate in approximately 80 per cent of the cases. Multiple factors determined the employability of these subjects. Emotional factors, particularly undue apprehension and anxiety, were exhibited by most subjects. The vocational needs were diverse, and in individual cases were multiple. Almost half needed significant vocational guidance.

During the interval of follow-up, an appreciable mortality rate occurred and was correlated, rather well, with the initial estimate of the functional and therapeutic capacity. Nevertheless, twice as many subjects either remained the same or improved as deteriorated, from both the cardiac and the employment standpoint.

The Cleveland Work Classification Clinic has helped patients with heart disease obtain employment, retain their jobs, and to be re-assigned. Over 50 per cent of the cases orig-

inally unemployed at the initial visit obtained employment. At the follow-up visit, 70 per cent of all the living subjects were employed.

The wide variety of jobs held by the subjects with heart disease dispels the concept of a *cardiac job*. To date, there has been no evidence that recommended employment of the cardiac subject is dangerous to himself or his employer. There have been no compensation claims.

BIBLIOGRAPHY

- 1 HELLERSTEIN, H. K., and GOLDSTON, E. Rehabilitation of patients with heart disease. *Postgrad Med* 15:265, 1954.
- 2 SHELDON, W. H., DUPERTUIS, C. W., and McDERMOTT, W. *Atlas of Men. A Guide for Somatotyping the Adult Male at All Ages*. New York, Harper & Bros., 1954.
- 3 FORD, A. B., and HELLERSTEIN, H. K. Energy cost of the Master two-step test, *JAMA* 164:1868, 1957.

CHAPTER 39 WORK CLASSIFICATION UNIT SURVEY

Sidney S. Sobin

The material in this report is based upon information obtained by means of a questionnaire that was submitted to 35 Work Classification Units in the United States. Twenty-five units replied to the questionnaire. One of them characterized itself as a diagnostic clinic, two units did not provide data for this study, three units were just beginning their work or were being organized, and three other units were inactive. A number of major work classification units were not included in this survey, their experiences are being presented at this Conference.^{1,2,3,4} This report consists, then, of the results obtained from 16 units.

It is granted that questionnaires may be influenced in their construction by the bias of the questioner. Often he may seek to affirm or deny his own experiences or opinions. This was patently true of the questionnaire used in the present study. However, certain questions were drawn up to collect factual material and other questions were designed to reveal the absence of certain data. A survey of this type can reveal *attitudes* only rarely but the nature of the reply as well as the data it

furnished, more often than not, provided an over-all impression of the unit's operation.

The data collected from 16 active units have been grouped in 11 major headings, as far as the replies make this possible (Tables 39-1 to 39-11). Several specific questions are also included in some of the tables. These include: (a) the number of patients with electrocardiographic abnormalities, (b) the relative value of various parts of the examination, (c) the value of the unit in promoting rehabilitation, and (d) the use of the unit in teaching. In view of the wide differences in purpose and manner of operation among the various units, specific summaries of most of the data cannot be drawn. Comment will be made upon trends that appear in the major categories.

Unit Characteristics

The character of the various units is shown in Table 39-1. Nine of the units were service in type (S), six were for pilot or demonstration purposes (P), and one was limited to research (R). A total of 2,130 new patients were studied in all units and there were 564

TABLE 39-1 CHARACTER OF UNITS

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Unit characteristics																
Nature of unit																
Starting date	10/54	12/54	8/54	9/55	9/56	9/52	1952	3/55	3/55	1952	7/56	6/51	9/55	9/54	8/53	3/52
Total number new patients	167	125	182	24	45	182	208	14	7	50	53	930	55	25	18	45
Total number revisits		25	83	0	68		12	2	7		10	864	27	0	0	13
Revisits in 1956	51	7	80	0	68		10	2	7				14	0	0	
Routine re-examination	6 mo	6-12 mo	6 mo	3-6 mo	3-6 mo	3 yr	no	6 mo	6 mo	3-6 mo	6 mo	6 mo	6 mo	6 mo	6 mo	6 mo
Operational days	2/wk	1/wk	1/wk	2/mo	3	2/mo	1/wk	2/wk	1/mo				1/wk	2/mo	2/mo	2/mo
Total operational days	325	106	81	24			28	28	7				84	61	12	1
Operational days 1956	120	40	48	16		22	40	28	5			230	46	27	1	
Av number new patients per day	2	2	2	1		1	2	1	1-2	3	4/wk	1	1	1		
Av number revisits per day	2	1	2	0		2		1	1-2	2	30/wk	1-2	0	0		
Location	OPD	Rehab	OPD	Rehab		OPD	OPD	OPD	OPD	OPD	OPD	OPD	OPD	OPD	HA	OPD

Legend: P, pilot, R, research, S, service, A, on referral only, B, indefinite, but usually only on referral, OPD, outpatient department, HA, Heart Association Code 1, Alameda County H A, 2, San Francisco H A, 3, Los Angeles County H A, 4, Greater Peoria H A, 5, Iowa H A, 6, Massachusetts H A, (Western), 7, New Jersey H A (St Michael's), 8, University of Buffalo, New York, 9, Dutchess County H A, 10, Westchester County H A, New York, 11, NW Ohio H A, 12, W Pennsylvania H A, 13, E Tennessee H A, 14, Galveston District H A, 15, Corpus Christi District H A, 16, Washington D C, H A

new patient examinations in 1956. Re-examination at chosen intervals was specified by most of the units. The frequency with which units met varied greatly; some units met daily whereas others met only on request. The majority met biweekly to semimonthly. From one to three new patients were examined at each meeting in the average unit. Most of the units operated in the outpatient department (OPD) or clinic of the hospital or medical school with which they were associated.

Referral Source

The sources from which the patients were referred varied considerably (Table 39-2). In some instances the sources were highly diversified with patients coming from private physicians, clinics, industries, unions, public

agencies, or being self-referred. A small number of work classification units operated primarily for the State Department of Employment or the Bureau of Vocational Rehabilitation (BVR).

Occupational Classification at Referral

The data regarding the classification of the patients at the time of referral to the various units are too incomplete to allow anything more than a general impression (Table 39-3). It may be seen that in the 10 units submitting information that could be classified there was a broad spread through the major job classifications, with some emphasis upon the skilled, semiskilled, and unskilled categories. Patients from the professional, managerial, clerical, or sales occupations were much less numerous. In certain

TABLE 39-2 SOURCE OF REFERRALS

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	R
Referral source																
Private physician	59	72	48		45	2	1	1	4		x	772	22	14	31	
Industrial physician	8	0	85		0		35	1			x	84	3	4	1	
Clinic	0	38	0		0		2	0		x			4	18	1	
Industry	6	0	0		0		0	1					1			
Union	2	0	0		0		0						13		1	
Self	51	0	0		0		0									
Public agency									1			74				
Public assistance	1	0	0	0	0	0	0									
BVR	1	14		24			170	11	3				24		1	
Dept. of Employ.	30	0								x						
School	5	1														
Social agency	5															

Legend: C, University Rehabilitation Department, x, referral source but other data not available, BVR, Bureau of Vocational Rehabilitation.

TABLE 39-3 OCCUPATIONAL CLASSIFICATION

Unit number	2	3	4	5	8	9	13	14	15	R
Occupational classification at referral										
Professional and managerial	4	19	2				1	1	1	
Clerical and sales	14	14	7			1	3	2		
Service occupations	8	4	0			x	2	4		
Agric., fishery	0	0	3	45		x		1		
Skilled	63	53	6			x	14	2	10	
Semiskilled	9	23	5			x	8	3	2	
Unskilled	27	20	2				15	10	5	1
Students			1					9		

TABLE 39-4 ELIGIBILITY

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Eligibility criteria																
Diagnosis lit disease	+	+	+	-	+	+	+	-	+	-	suspect	+	-	+	-	-
Doctor consent		+	+	+	+	+		+	+	+	+		+	+	+	+
Employed at referral	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-
Pre-employ. exam.	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-
In compensation litigation	-	-	-	-	-	-	-	-	+	-	+	-	-	+	-	-
Acute illness	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-
Financial limits	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
Charge	-	-	-	BVR	-	-	BVR	agency	-	-	-	-	-	-	-	-

50

TABLE 39-5 EXAMINING GROUP

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Examining group																
Internist-cardiologist	+	+	+	+	+	+	+	+	+		+	+	+	+	+	+
Psychiatrist	-	-	-	-	-	-	-	-	+		-	+	-	-	±	-
Rehabilitation	-	+	-	+	+	+	+	+	+		-	-	+	+	+	±
Placement	-	-	-	-	-	+	+	+	+		-	-	+	-	+	±
Job analyst	-	+	-	+	±	+	-	+	+		+	+	-	-	+	±
Technician	-	+	+	+	-	-	-	-	-		-	+	-	-	-	-
Psych social worker	-	-	±	-	-	-	-	-	-		-	-	-	-	-	-
Psychologist	-	-	-	-	-	-	-	+	-		-	-	-	-	-	-
Social worker	+	+	+	+	±	+	+	-	+		+	+	+	+	+	+

units with special interests or location, all the patients were in a single occupational classification, as in Unit No 5 in this table in which all the patients were in agriculture

Eligibility Criteria

A diagnosis of heart disease at the time of referral was necessary in only nine of the units (Table 39-4). The consent of a physician was required in 12 of the units. Only one unit required that the patient be employed or have a job to which he could return. In a few instances the patients were eligible only if recovered from acute illness or if there was no involvement in workman's compensation litigation. In no instance did the unit charge the patient for services and no patient was excluded by financial limitations of the clinic itself.

Components of Examining Group

The staff of the units responsible for examining and evaluating the patients varied considerably from unit to unit (Table 39-5).

All units included an internist or cardiologist. There was no common pattern as far as the rest of the personnel was concerned. From the designations shown in the chart, it is seen that almost any combination of staff may be selected.

Components of Examination

The various units were consistent as far as the standard examining procedures were concerned, including the history, clinical examination, electrocardiographic and fluoroscopic examination of the heart (Table 39-6). Most units also carried out an examination of the blood and urine. Eleven units used the step test to elicit electrocardiographic abnormalities suggesting coronary insufficiency and one unit used standardized work on a treadmill. Pulmonary function studies were done rather infrequently. The laboratory procedures and the referral data were considered adequate in the majority of units making this information available.

TABLE 39-6. EXAMINATION PROCEDURES

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Adequate referral data	+	-	-	+	-	+		-	+		-	+	+	+	+	+
Examination components																
Data by patient	-	-	+	+	+	+	+	-	+		+	-	-	+	-	+
Social history	+	+	+	+	+	+	+	+	+		+	+	+	+	+	+
Psychiatric	-	±	+	-	-	-	-	+	+		-	+	-	-	-	-
Job analysis	+	+	+	+	+	+	+	+	+		+	+	+	-	+	+
Work history	-	+	+	+	+	+	+	+	+		+	+	+	+	+	+
Medical and cardiac	+	+	+	+	+	+	+	+	+		+	+	+	+	+	+
EKG	+	+	+	+	+	+	+	+	+		+	+	+	+	+	+
Fluoro	-	+	+	-	±	+	+	+	+		+	+/V	+	+	+	+/B
X-ray	-	-	-	+	+	+	+	+	+		-	+	+	+	+	±
Blood	-	-	+	+	+	±	+	+	+		-	+	+	+	+	±
Urine	-	-	+	+	+	±	+	+	+		-	+	+	+	+	+
Exercise test		S	T	S	S	S	-	S	S		S	S	S	-	S	S
Pulmonary function		-	+	-	±	-	-	+	+				-		-	
Adequate lab procedures	+	+	+	-	+	+	+	+	+		-	+	-	+	-	+

Legend +, present, -, absent, ±, done irregularly, S, step test, T, treadmill test, +/V, vectorcardiogram, +/B, ballistocardiogram

TABLE 39-7. CHARACTERISTICS OF PATIENTS

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Patient characteristics																
Age distribution	15-60	20-61	20-74	21-61	32-77			20-60	16-65		15-66	18-35	17-69	22-67	20-65	22-70
Average	50	50+	46-50	42	50			20-40	45-55		47	35-54	40	50	50	46
Sex Male		110	131	23	42			170	12	5		49	82%	48	21	17
Female		15	2	1	3			38	2	2		4	18%	7	4	1
Dependent family size			2+		3				4				3			
Education Grade		50	44	16	29			7	5					10	9	50
High school		12	65	4	15			6	2					9	8	1
College		8	20	4	0			1	0					6	1	10
Professional		2	4	0	0			0	0					0	0	1
Years employment present job																
5 years			12		15			4	3					10	8	22
15+			6		30											
Duration of cardiac illness																
0-6 months		3			4			3				5			2	1
6-12 months		11			1			4	2					4	5	
Over 12 months		47			40			7	5					16	15	7
Hospitalization due to heart disease				81		13		7						1		
2 mo																
Work days lost by illness																
Compensation litigation																
Before WCU exam			3	0										1		
After WCU exam				2										1		

Characteristics of Patients

The patients studied in all the units were similar in character (Table 39-7). The age range was broad, with a mean between 40 and 50 years. Males were in a strong majority in all units. In the responses to the questionnaires, the information concerning the size of the family of dependents, the education of the patient, his years of previous employment, and the details of his cardiac illness

and its impact upon his employment was too meager to permit any broad conclusions. The information that the survey yields is included in Table 39-7.

Unit Diagnosis

The types of heart disease encountered in the various units were similar except for the regional differences in the incidence of rheumatic heart disease, which were anticipated

(Table 39-8). In most of the units, 10 to 20 per cent of the patients referred for study were found to have no evidence of heart disease. One third to two thirds of the patients seen in the various units had coronary artery disease. This high incidence of coronary ar-

teriosclerosis is felt to be a reflection of the age level of the patients studied.

Unit Reports

In nearly all the units, the reports were in narrative form (Table 39-9). Form letters

TABLE 39-8 FINDINGS

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Unit findings																
Total number new patients	76	125	133	24	45		208	14	7		53	930	55	25	18	45
No heart disease	7	4	30	6	7		26	3				197				9
Hypertension	2		2	2	6		6	1				32				1
Arteriosclerotic heart disease	41	81	74	13	27		88	5	5			308	24	12	11	14
Angina pectoris				2	16								12	3	8	
Myocardial infarction				8	11								12	9	3	
Rheumatic heart disease	12	33	6	5	7		65	3	2			210	21	6	3	8
Hypertensive c-v dis.	10	4	9	2	2		28					6	3	5	2	9
Luetic												5		1		2
Congenital	4	3	1		1		11	1				12	6	1	2	
Ht dis with pul dis				1	2				1			36	2	0	0	
Pulmonary dis. without heart disease			1									14				
Other		14		1			1	1				177				2
EKG abnormality																
Total					27			2	5					25	18	

TABLE 39-9 UNIT ROUTINE AND EMPHASIS

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Of most value in examination																
History			+	+	+	+	+	+	+		+	+		+	+	+
Physical examination						+	+	+	+					+		
Patient conference																
Routinely held	+	+	+	+	+	+	+	+	+		+	+	+	+	+	+
All personnel	+	+	+	+	+	+	+	+	+		+	+	+	+	+	+
Patient attends	-	-	+	+	+	-	+	-	+		-	-	-	-	a	-
Patient informed WCU rec																
By letter															+	
By own physician	+	+	+			+	+				+					
By WCU			+	+	+	-	+	+	+		-	+	+	-		+
Unit report																
Type of report																
"Form"									+			b		+		
Check list	+C						+	+								
Narrative letter	+	+	+	+	+	+	+	+			+	+	+	-	+	+
Phoned			+													
Sent to																
Referral source	C	+	+	+	+	+	+	+	+			+	+	+	+	+
Private physician	+	+	+	+	+	+	+	+	+			+	+			
Industrial physician			+				+		+d			+	+			
Does WCU promote rehabilitation?	+	+	+	+	+	+	+	+	+		+		+	-	-	
Is unit used in teaching?	-	+e	+f	-	-	-	+	-	-		-	+f	-	-	-	+

Legend a, irregularly, b, now abandoned, C, work capacity report only, d, sent only at patient's request; e, unit integrated in social work and rehabilitation teaching, f, postgraduate medical teaching.

and check lists were used uncommonly. Physicians tended to prefer a comprehensive narrative report, whereas the various agencies showed some preference for a more simple report such as a form letter or a check list. In the majority of the units the history was considered the most important segment of the examination. All but two of the units felt that the work classification unit helped to promote rehabilitation. Five of the 16 units were used in some form of teaching.

Cost of Operation

Seven units supplied information regarding the cost of their operation (Table 39-10). It is not clear whether these reported costs also included less apparent items such as the cost of clinic, office space and maintenance, and supplies, postage, and other similar materials. Although it would be highly desirable to know the cost of the operation of the classification unit per patient, the data resulting from this survey do not make available a reliable figure.

Comparison of Work Status on Referral and Unit Recommendations

An effort was made to compare the working status of the patient at the time of his referral to the unit with the recommendations of the classification unit and its follow-up data. In many instances the informa-

tion made available was incomplete. Nearly half of the units reporting did not provide follow-up information relative to their own work recommendations. Nine units did provide sufficient information to draw up a table that is of some interest in the matter of *reclassification* (Table 39-11). It is clear that a significant percentage of the patients studied, who were unemployed because of heart disease, or who were on restricted programs of work, were recommended to full work programs.

In the original questionnaire references were made to duration of illness, duration of unemployment, and follow-up recommendations. These questions have been deleted from the table since almost no replies to these questions were obtained.

Conclusions

The tables are intended to show only a summary of the information obtained. The survey does permit certain conclusions pertaining to the operation of nearly all the work classification units that responded to the questionnaire. It is clear that much needs to be done to obtain a simple factual account of the work of the various units, to provide complete and accurate, comprehensive data concerning the patients examined, to make available some means of evaluating and validating their work recommendations, to establish consistent and uniform methods

TABLE 39-10 COSTS IN DOLLARS

Unit number	1	2	3	4	5	6	7	8	9	10	11	12	13	16
Cost of WCU operation														
Total yearly		8900+	11000		7500	4500		12000				27825	4700	
Medical director		100 mo	200 mo		paid				paid			special		
Cardiologist	25 sess mo	25 sess paid	100 mo 300 mo	paid		vol	vol	2 @ 3000	12 50 sess paid	50 wk 10 wk	research	paid		
Social Worker	205 mo			paid		paid					budget	72 50		
Secretary			65 mo		paid	paid		3000			with	108		
Technician		paid	100 mo no budget	paid		paid		3500			full	30		
Other costs (lab, etc.)						budget			hosp cont.		staff			
Vocational counselor	210 mo	paid		paid		paid								
Rehab counselor									paid					50 (R.V.)

TABLE 39-11. REFERRAL WORK STATUS AND WCU RECOMMENDATION

Unit number	1	2	3	4	5	8	9	12	13	14	15	
UNEMPLOYED *	46	59		12	7		7	4	45	14	20	14
WCU recommendation												
Sick leave		0			3					1		
Restricted work		47-5NF			4	3	4			15		14-2NF
Full work		8-2NF		12		4				4		
EMPLOYED	18	13	129	12	37	7	3	148g		5		4
Employed, on sick leave		13	49			6				1		1
WCU recommendation												
Continue leave			7							1		
Restricted work		12	16			3						1-NF
Full work		1	26			3						
Employed, restricted work			30	1	22		2			1		3
WCU recommendation												
Sick leave			2		5-4NF							
Restricted work			11	1	17		2			1		3
Full work			17									
Employed, full work			50	11	15		1			3		
WCU recommendation												
Sick leave			1									
Restricted work			9		6-3NF					2		
Full work			40	11	9		1			1		

Legend g, total of 193 patients (45 unemployed, 148 employed), the work recommendation relative to referral work status was 87, same, 90, restricted work, 4, increase, and 12, retired. NF, not followed

* Unemployed owing to heart disease

of recording, collecting, and reporting observations and results, and, finally, for determining actual costs of operation

It is not the purpose of this report to make critical comments concerning the various work classification units. It is clear that most of the units reporting provide a definite service to the community in which they function and, generally, a service not available elsewhere. If the specific needs of each community are met, the existence of the unit is justified. However, it is apparent that in many instances a better total job can be done, not only in the technique of operation but in providing a better educational and demonstration program for many medical and nonmedical segments of the community. In most communities there seems to be a continuous need to say and to demonstrate that individuals with heart disease can work

BIBLIOGRAPHY

- 1 BRONSTEIN, L. H. Experience of the Work Classification Unit at Bellevue Hospital. First Wisconsin Conference on *Work and the Heart*, New York, Paul B Hoeber, Inc., 1959, Chap 35
- 2 CLARK, R. J. Experience of the Cardiac Work Classification Unit of Boston, Massachusetts. First Wisconsin Conference on *Work and the Heart*, New York, Paul B Hoeber, Inc., 1959, Chap 36
- 3 GELFAND, D. Experience at the Cardiac Work Classification Unit of the Heart Association of Southeastern Pennsylvania (Philadelphia). First Wisconsin Conference on *Work and the Heart*, New York, Paul B Hoeber, Inc., 1959, Chap 37
- 4 PARRAN, T. V., et al. Results of Studies at the Work Classification Unit of the Cleveland Area Heart Society. First Wisconsin Conference on *Work and the Heart*, New York, Paul B Hoeber, Inc., 1959, Chap 38

CHAPTER 40 EXPERIENCE WITH CARDIACS AT THE ALTRO WORKSHOPS*

Abraham Jezer

HISTORICAL BACKGROUND AND SETTING

The Altro Health and Rehabilitation Services has had over 40 years of experience with the rehabilitation of tuberculous patients and for more than eight years has been concerned with rehabilitation of patients with cardiovascular illness. In 1954 it began accepting patients recovered from mental illness.

The major occupation provided is at the single-needle power sewing machine. This is

sedentary work, demanding little physical energy output. It is possible for almost anyone with some manual dexterity, and from dull normal intelligence up, to learn the simplest sewing operations in a reasonably short period of time. Payment for work produced is on a piecework basis at the highest rates set for similar processes in unionized industry in the city of New York. When the patient can sew a straight seam, he is given simple garments to work on, and he begins to earn for his production.

Obviously, even if the new Altro employee were skilled at the sewing machine, his limited number of hours of work would lessen his productive income. For most of the patients, for many weeks earnings fall far below the income needed for themselves and their families. Therefore, the Altro Health and Rehabilitation Services subsidizes the earnings up to the decent minimal budget requirements for the family. If the patient is eligible for public assistance, it is expected

* This investigation was supported by grants from the National Heart Institute, the United States Public Health Service (Contract Number SApH 59478), and the New York Heart Association. Studies were made together with the Evaluative Study Staff including B. J. Black, Administrator; Daniel Adelson, Social Psychologist; Florence Hasellorn, Casework Supervisor, Leopold Bellak, M.D., Psychiatrist; Gloria Auerhan, Caseworker; Irwin Friedman, Caseworker, Hannah Chrystall, R.N., Nurse, Annabelle Replansky, Jr., Research Assistant, and Brunah Szapira, Marian F. Loew, M.D., Shirley Madonick, and Libby Eigen, Staff Assistants.

that this, too, will be drawn upon. Many patients reach a point of full self-sufficiency before they leave Altro Workshops, earning as much as they may do later in the garment trade outside or in other occupations more suited to them.

It should be pointed out that, over the years, careful studies of both the tuberculous and cardiac experience have shown that only 15 to 17 per cent of Altro graduates have actually gone into the garment operating trades. The vast majority are rehabilitated folk, who have returned to or gone into a wide variety of other trades and occupations. In a very real sense, Altro has been, for most of its graduates, a transitional experience to normal vocational living.

During the past two years, the Altro Workshops have added two new programs, one is a classroom in office and business machines practice; the other is a work-study program in sewing machine repair. These are keyed into the factory operations in such a way as to lead naturally from a classroom setting to job testing, to preparation for job placement.

DESCRIPTION OF ROUTINE FOR CARDIAC PATIENTS

At the time of application, all previous medical, social, and vocational history is secured to determine whether the patient requires the services provided by Altro and can benefit from them. After examination by the cardiologist and an interview with the patient and his family by the social worker, the patient is started usually on a schedule of three or four hours of work. The patient visits the Workshops in advance of his acceptance. During the period of rehabilitation at the Workshops, he is re-examined periodically by the doctor, and seen by the social worker as often as his condition requires.

HOURS OF WORK The patient spends the full working day at the Workshops, but works only the hours prescribed by the physician to the Workshops. The patient completes half of his daily assignment, has

luncheon, and rests two or more hours. Then he repeats the morning procedure and leaves for home at 5:00 P.M. In some instances, if the patient has a long ride home, he may leave earlier. As his hours of work are increased, the rest period is shortened until he has only one hour for luncheon. A station wagon transports the cardiac patient to and from the nearest bus or subway station.

SELECTION OF TRAINING In an industrial plant, workers are selected because of skills for the work to be performed. At Altro, they are selected because they have physical limitations, require carefully graduated work, have not worked for many months or years, and fear any physical activity, often believing they should do less work than the doctor recommends. The manager of the Workshops is given the patient's work history, vocational objectives, the doctor's recommendation, particularly as to whether the patient may require sheltered employment for a long or indefinite period. With this information, subsequent observation, and discussion, the manager decides which patient shall receive intensive trade training and which is to be taught one or more simple operations. Trade union rates are paid on a production basis from the moment the patient is able to sew a straight seam and his work can be used.

RETURNING TO WORK The length of stay at the Workshops is determined by the patient's medical and psychological progress and his work objective. If he plans to return to his old job and this is not contraindicated, he is advised to stay until he has demonstrated his ability to work the number of hours that his old job entails. If his interest is to return to school for educational or vocational training, the doctor usually permits graduation from the Workshops when the patient has reached a level of five to six hours' work, if he believes that at the completion of the chosen training the patient will be ready for normal work. Housewives are also served at the Workshops.

AGE ON ADMISSION AND CRITERIA OF ADMISSION. Patients are admitted between the ages of 18 and 60. Exceptions have been made when former employers agree to reemploy

patients. Unfortunately, the available space at the Workshops has limited the number of patients who, because of the chronicity of their disease, cannot be expected to return to industry. However, there is no age limitation among the limited number of cardiacs in the *long term or permanently sheltered* category, at present making up 20 per cent of the total capacity. Patients up to and including Class III C, according to the functional classification of the New York Heart Association, are accepted.

FACTORS ASSOCIATED WITH SUCCESS AND FAILURE IN REHABILITATION

The question which, in a sense, touched upon or embraced all the purposes of the project as originally outlined was "What factors are associated with success and failure in the rehabilitation of the disabled cardiac?" To answer this question, two separate groups of patients who were served at the Altro Workshops at different periods of time were analyzed. These included 144 served from the opening of the Workshops for the cardiac disabled in 1948 through September, 1953, and 58 patients served from October, 1953, through August 31, 1955.

The two studies were done separately. The analysis of the first group was concerned with four areas: (a) medical, (b) social-psychological, (c) experience at Altro Workshops, and (d) work history and education. One hundred thirty of the 144 patients were also rated on the rehabilitation level by the supervising caseworker. These were the patients who had completed the period of rehabilitation at Altro as of September 30, 1953, including those who had become *outsiders*, the workers who form the regular staff of the Altro Workshops or *permanently sheltered*, i.e., those who are able to continue a productive work life only while in the special environment provided by Altro, and have been accepted on this basis. The analysis of the 58 patients served from October, 1953, through August, 1955, was not quite as comprehensive and did not include ratings on the rehabilitation level. A mailed ques-

tionnaire provided the follow-up in contrast with the personal interviews that were used for the original group of 144 patients.

Cases of 144 Cardiac Disabled Served at the Altro Workshops

Follow-Up

While periodic follow-up from 1948 through September, 1953, chiefly via mail, had been made from the beginning of the patients discharged from the Altro Workshops, for the present study a more comprehensive follow-up questionnaire was prepared and as many of the patients as possible were seen in a personal interview. In addition, a follow-up questionnaire for physicians was used. On the basis of this follow-up, a judgment by the supervising caseworker was made as to whether or not the patient was a success or a failure in accordance with predetermined criteria.

Record Analysis and Coding

The medical, casework, Altro Workshops, and other records, follow-up questionnaires, and other available material were analyzed and a profile on each patient was prepared on the basis of the over-all code. Subsequently, *success or failure* was related to the various factors in each of four areas: (a) medical history and history of cardiac illness, (b) history of work experience and education, (c) history of experience at the Altro Workshops, and (d) psychosocial indicators.

Over-all Code

The final code evolved from a variety of sources. It came primarily from those persons who had worked closely with the cardiac program at Altro since its inception in February, 1948, including the cardiologist, the psychiatrist, the administrative head of the project, the casework supervisor, the social psychologist, and the members of the research staff. Schedules and questionnaires

(b) the questionnaire used in the York

Study of Mental Health (New York Hospital); (c) the Massachusetts General Hospital Study; (d) Dr Leopold Bellak's classification of "Reactions to Illness."

General Description of Cases

SEX, NATIVITY, AND ETHNIC BACKGROUND. Of the 144 clients, the vast majority, 124 or 87 per cent, were male and 29 or 13 per cent were female. The large number of male, foreign born, white, is notable (Table 40-1).

TABLE 40-1 DISTRIBUTION OF 144 CARDIAC PATIENTS BY SEX, NATIVITY, AND ETHNIC BACKGROUND

<i>Sex, nativity, and ethnic background</i>	<i>Number</i>	<i>Per cent</i>
All patients	144	100
Male, native born white	54	37
Male, foreign born white	71	50
Female, native born white	11	8
Female, foreign born white	8	5

AGE ON ADMISSION TO ALTRO Ninety-six patients or 66 per cent fell in the age ranges 36-45 and 46-55. Twenty-five or 18 per cent fell in the age ranges below 36, 22 or 15 per cent in the 55-60 age range. Only one client was in the 61-65 group (Table 40-2).

TABLE 40-2 DISTRIBUTION OF 144 CARDIAC PATIENTS BY AGE ON ADMISSION

<i>Age on admission</i>	<i>Number</i>	<i>Per cent</i>
All ages	144	100
18-25	11	8
26-35	14	10
36-45	43	30
46-55	53	36
56-65	23	16

EDUCATION Of the 105 clients for whom information as regards education was available, 43 or 41 per cent had completed elementary school, 29 or 28 per cent had some high school, 15 or 14 per cent had completed high school, and only 9 or 8 per cent had gone beyond high school.

MAJOR OCCUPATION The clients came from

many occupations (Table 40-3). Twenty-seven patients or 19 per cent came from the garment operating and associated trades. This was followed in frequency by the factory, electrical, mechanical, and building industry workers who made up 24 or 17 per cent. Only 9, or 6 per cent, fell into the professional or managerial category. This distribution follows the general occupational character of our population.

TABLE 40-3 OCCUPATION BEFORE AND AFTER TRAINING AT ALTRO WORKSHOPS

<i>Major occupation</i>	<i>Number before</i>	<i>Number after</i>
Professional and managerial	9	2
Clerical	6	15
Sales	12	6
Service	7	9
Agricultural	4	0
Truck, bus, and taxi drivers	11	1
Garment operating and associated trades	27	20
Business owners	13	4
Butchers, bakers, chefs, food industry workers	12	4
Factory, electrical, mechanical, building industry workers	24	10
Miscellaneous occupations (housewives)	12	4
No work history	0	36
Major occupation not known	1	16
Attending school	—	2
Total	144	129*

* Fifteen still in rehabilitation program at the Workshops at the time of the study.

STABILITY OF WORK HISTORY BEFORE ONSET OF ILLNESS The majority of clients, 111 or 78 per cent, had fairly stable work histories and were classified as having had regular full-time work. Only 13 or 12 per cent were placed in the irregular employment category (Table 40-4).

MARITAL STATUS AND CURRENT FAMILY STABILITY Of the 144 clients, there were 92 or 64 per cent widowed, 27 or 19 per cent single, 5 or 3 per cent divorced; and 9 or 6 per cent separated. Seventy-four or 52 per cent were rated as having good current family stability, and 37 or 25 per cent as having

TABLE 40-4. DISTRIBUTION OF CARDIAC PATIENTS BY STABILITY OF WORK HISTORY BEFORE ONSET OF ILLNESS

Stability of work history	Number	Per cent
All patients	144	100
Regular full-time employment	111	78
Irregular employment	18	12
Attended school and had never worked	6	4
Housewives	3	2
No information	6	4

adequate current family stability, and 32 or 22 per cent as having poor current family stability.

Most of the Altro clients come from the lower and lower-middle socioeconomic strata. This judgment is based on our knowledge of their educational and occupational backgrounds, as well as their incomes and places of residence.

Since in this group Altro was selective with respect to age, functional classification, and other factors, we do not know whether the percentage of those in the various age ranges or in the functional classifications really reflects the community need.

TABLE 40-5. DISTRIBUTION BY FUNCTIONAL CLASSIFICATION

Functional classification	Number	Per cent
All patients	144	100
I B	21	14
II B	4	3
I C	6	4
II C	87	60
III C	23	16
III C-III D	1	1
II D	1	1
Not established	1	1

FUNCTIONAL CLASSIFICATION If functional classifications as determined by the criteria of the New York Heart Association are grouped together thus: *best* including I B, I C, and II B; *middle* including II C; and *poorest* including III C and III D, we find that 87 or 60 per cent fall in the *middle* group, while 31 or 22 per cent fall in the *best*, and 25 or

17 per cent in the *poorest* classifications respectively (Table 40-5).

ETIOLOGY: As regards etiology, 92 patients or 64 per cent had arteriosclerotic or arteriosclerotic and hypertensive heart disease. There were too few instances of hypertension as a cause for disability from heart disease to provide a separate statistical analysis; therefore the seven patients in this etiological classification are included here. In addition there were six or 4 per cent who were classified as functional neurosis as well as arteriosclerotic, or arteriosclerotic and hypertensive heart disease. There were 43 or 30 per cent who had rheumatic heart disease (Table 40-6).

TABLE 40-6. DISTRIBUTION BY ETIOLOGY

Etiology	Number	Per cent
All patients	144	100
Rheumatic heart disease	43	30
Arteriosclerotic and/or hypertensive heart disease	98	68
Congenital heart disease	2	1
TB pericarditis	1	1

Analysis of Factors Associated with Success and Failure

Examining the factors related to success and failure in each of the four areas under consideration, this report will give, chiefly, the findings for those factors that appear related, although many additional factors were studied, as can be seen by the code for each of the four areas. Of the 130 clients who were rated on a rehabilitation level, 60 or 46 per cent were considered fully rehabilitated; 25 or 19 per cent were considered partially rehabilitated; and 45 or 35 per cent were rated failures. Fourteen clients were not rated on a rehabilitation level either because they were still in the process of rehabilitation at the Workshops or an insufficient period of observation had elapsed.

Medical Factors

The most striking, if not unexpected, factor related to success and failure in the re-

rehabilitation of the cardiac disabled is functional classification (Table 40-7)

It is seen that there was a definite trend for the better functional classifications to be associated with success. If Classes I B, I C, and II B are grouped together as the *best* functional classification, we find that 26 or 93 per cent were either successes or partial successes. Fifty-one or 65 per cent of the II C's or *middle* functional classification

TABLE 40-7 RELATION OF FUNCTIONAL CLASSIFICATION TO SUCCESS AND FAILURE IN REHABILITATION

Functional classification	Success		Partial success		Failure	
	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent
I B, I C, II B	20	71	6	24	2	5
II C	37	47	14	18	28	35
III C, III D	3	14	5	12	15	64
Total	60		25		45	

were successfully or partially rehabilitated. Finally, the III C group or *poorest* functional classification showed only eight or 38 per cent successful or partially successful in rehabilitation. It appears that the chances for a successful rehabilitation after a transition experience at Altro are very high if the cardiac function permits. On the other hand, if the physical condition is very poor, chances of successful rehabilitation are rather small. Indeed, three of the eight patients in the *poorest* functional classification who were considered successfully rehabilitated were either permanently sheltered or employed at Altro. Functional classification in itself does not, of course, provide a complete yardstick as to potential for success and failure. Particularly does it fail to differentiate the patients in Class II C. It also fails to account for failures in the *best* and successes in the *poorest* functional classifications.

LENGTH OF ILLNESS AT TIME OF ADMISSION TO ALTRO. The next most striking, if not unexpected, finding was the way in which length of illness at time of admission sep-

arated the successes from the failures (Table 40-8)

There was a definite tendency for those who had been ill for a long period of time to fall into the failure column. If the patients shown in Table 40-8 are divided into two groups, we find that in those who were ill from 0 to 2.9 years, 46 or 75 per cent were successfully rehabilitated, whereas in three or 5 per cent rehabilitation was partially successful, and 12 or 20 per cent were failures. In the group of patients who had been ill for 3 years or more, rehabilitation was successful in only 13 or 22 per cent, partially successful in 17 or 29 per cent, and a failure in 29 or 49 per cent.

TABLE 40-8 RELATION BETWEEN LENGTH OF ILLNESS AT TIME OF ADMISSION AND SUCCESS AND FAILURE IN REHABILITATION

Length of illness	Success		Partial success		Failure	
	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent
0-5.9 months	7	100	—	—	—	—
6-11.9 months	16	84	1	5	2	10
1-1.9 years	17	63	2	7	8	30
2-2.9 years	6	75	—	—	2	25
3-3.9 years	1	11	4	44+	4	44+
4 years and over	12	21	18	31	29	48
Total	60		25		45	

When length of illness and functional classification are taken together, we have an even more powerful tool for sorting out successes from failures. This is more striking for the patients in Class II C than for those in the *best* and *poorest* classifications. With respect to the II C functional classification, 35 of 44 or 80 per cent of those in the 0-2.9 years category were either fully or partially rehabilitated, as contrasted with 14 of 31 or 45 per cent of those unemployed for 3 years or more.

If the patients in the *best* functional classification are considered, 11 or 100 per cent of those who had been ill for 0 to 2.9 years were fully rehabilitated. This is contrasted with eight fully successful and three partially suc-

cessful rehabilitations in 11 of 12 or 91 per cent of patients in the same functional classification but who had been ill for 3 years or longer. Considering the patients in the *poorest* functional classification, it was found that three of six patients or 50 per cent of those ill for 0 to 2.9 years were fully rehabilitated, compared with no fully and five partially successful results in 16 or 31 per cent of individuals in the same functional classification but ill for 3 years or more. In each category there were a few clients for whom information was lacking.

TABLE 40-9 RELATION BETWEEN LENGTH OF ILLNESS AND LEVEL OF REHABILITATION IN THE II C FUNCTIONAL CLASSIFICATION

Length of illness	Success		Partial success		Failure		Total	
	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent	Num-ber	Per-cent
0-2.9 years	32	73	5	7	9	20	44	100
3-20 years and over	5	16	9	29	17	55	31	100
Total	37		12		26		75*	

* For 12 of the 87 in the II C functional classification, length of illness at time of admission could not be established.

Table 40-9 shows that length of illness also helps to anticipate full and partial successes or failures in the patients in the II C functional classification. Individual cases taking an unexpected direction still have to be explained. But taken together, functional classification plus length of illness would seem to offer a pretty good device for prediction. In comparison, such factors as sex, nativity, and ethnic background, age on admission to Altro, and etiology of heart disease do not in themselves appear strongly related to success and failure at the Altro Workshops. It was noted that there was a higher percentage of failures in those with rheumatic heart disease than in those with arteriosclerotic and hypertensive heart disease. Fully successful rehabilitation was achieved in only 13 of 35 or 37 per cent of patients with rheumatic heart disease as compared to 42 of 85 or 49 per cent of those in the arteriosclerotic or arterio-

sclerotic and hypertensive group. There were no failures among the seven patients classified as having functional neurosis (emotional disability) as well as rheumatic, arteriosclerotic, or arteriosclerotic and hypertensive heart disease. If age is considered separately, and distinguishing the cases according to etiology, it was found that there was less chance for successful rehabilitation in patients with rheumatic heart disease who were over 35 years of age and for patients with arteriosclerotic and hypertensive heart disease who were over 50 years of age.

Always keeping in mind the basic medical factors related to success and failure in rehabilitation, the variables within the other major sections of our code were considered next. Both *Work History and Education* and *Psycho-Social Factors* may be judged, at least to some extent, at the time of application, and finally at the *Altro Workshops Experience*.

Work History and Education

STABILITY OF WORK BEFORE ONSET OF ILLNESS. There is some indication that work stability before the onset of illness is related to success. There were 66 per cent successes for those who had regular full-time jobs before onset of illness.

The stability of work history before the onset of illness may help us better to differentiate the cases in Class II C. In this functional classification, of those with irregular employment there was only one full success as compared to two partial successes and four failures. Similarly, for those with irregular employment in Classes I B and I C there was only one full success as compared to three partial successes and one failure. The fact that so many of the Altro patients had regular full-time employment before the onset of illness makes this a somewhat less valuable index for prediction of results.

TIME WITHOUT WORK BEFORE COMING TO ALTRO. There appears to be a definite relationship between time without work before coming to Altro and success or failure. Of those without work for less than two years 49 or 58 per cent were successes, 18 or 21

per cent were partial successes; and 18 or 21 per cent were failures. In the group who were without work for from two to three years, six or 40 per cent were successes, two or 13 per cent were partial successes, and seven or 47 per cent were failures. When the individual had been without work for three years or longer, there were no full successes; two or 17 per cent were partial successes, and ten or 83 per cent were failures.

When all these facts are taken together, the time without work before coming to Altro appears to be a significant factor determining success or failure. The chance for rehabilitation of a patient in Class III C out of work more than a year and of a person in Class II C out of work more than three years appears very slim indeed. The length of time without work before coming to Altro is an indication of either very serious heart disease or a severe emotional disorder superimposed upon less serious heart disease. The severe organic II C clinically resembles a III C, and a less severe II C is lifted into the II C category by superimposed psychological factors. In this exploratory study, we are not attempting to analyze the contribution to the final result of such variables as length of illness. Obviously, they are related.

LEVEL OF EDUCATION There does appear to be some trend for better education to be associated with rehabilitation. Thus, only about 14 per cent of the group who had finished high school or had gone beyond were failures. This appears to be a slight trend and we will not explore it here.

In comparison with the factors mentioned above, such variables as major occupation and work after onset of illness (58 per cent of our clients did not work) seemed to have little or no relationship with successful rehabilitation at the Altro Workshops.

Psychosocial Indicators

MARITAL STATUS There appeared to be some tendency for divorced and separated men to be failures since this occurred in seven of ten, or 70 per cent (Table 40-10). It will be noted, also, that our two Classes I B and I C failures were single. In Classes

I B and I C there are no married failures. In contrast with those who were separated or divorced, 72 per cent of the married men and 60 per cent of the widowed men were either full or partial successes. Perhaps instability reflected in work experience as well as in marriage may be the common denominator for the failure to adjust successfully.

TABLE 40-10 MARITAL STATUS IN RELATION TO LEVEL OF REHABILITATION

Marital status	Success (Number)	Partial success (Number)	Failure (Number)	Total (Number)
Married	44	17	23	84
Widowed	5	1	4	10
Single	9	6	11	26
Separated	1	1	4	6
Divorced	1	—	3	4
Total	60	25	45	130

CURRENT FAMILY STABILITY There was a tendency for better current family stability to be associated with successful rehabilitation. When the functional classification was held constant and the rating on current family stability was examined in the group with the best functional classification, of those with good family stability ten or 83 per cent were successfully rehabilitated and two or 17 per cent were partially successful. When, for the same functional classification, current family stability was adequate, five or 71 per cent were fully rehabilitated and two or 29 per cent partially so. In those patients of the best functional classification whose current family stability was considered poor, rehabilitation was successful in five or 56 per cent, partially successful in two or 22 per cent, and a failure in two or 22 per cent.

When current family stability was reviewed in patients in the poorest functional classification, it was found that complete or partial rehabilitation was achieved in three of nine or 33 per cent of those with good family stability, four of nine or 44 per cent of those whose family stability was adequate; and only one of five or 20 per cent of those with poor family stability.

There is thus a suggestion that if the rating

is *poor* in current family stability, successful rehabilitation is less likely for those in either the *best* or the *poorest* functional classifications.

For those whose functional classification was II C, the ratings on current family stability appeared more clearly associated with success or failure. Thus, of those rated as having *good* family stability 39 of 48 or 81 per cent were rehabilitated (58 per cent fully and 23 per cent partially); eight of 17 or 47 per cent of those rated as having *adequate* family stability were rehabilitated (35 per cent fully and 12 per cent partially); and four of 14 or 29 per cent of those rated as having *poor* family stability were rehabilitated (22 per cent fully and seven per cent partially).

TABLE 40-11 ABILITY TO COPE WITH PREVIOUS LIFE STRESS AND FUNCTIONAL CLASSIFICATION IN RELATION TO REHABILITATION LEVEL

Functional classification	Ability to cope with previous life stress	Success		Partial success		Failure		Total
		Num- ber	Per cent	Num- ber	Per cent	Num- ber	Per cent	
Best	Good	7	88	1	12	—	—	8
	Fair	12	75	4	25	—	—	16
	Poor	1	25	1	25	2	50	4
Middle	Good	18	75	3	12+	3	12+	24
	Fair	18	45	7	17+	15	37+	40
	Poor	1	7	4	29	9	64	14
Poorest	Good	2	50	1	25	1	25	4
	Fair	1	7	3	21+	10	71	14
	Poor	—	—	1	20	4	80	5

Although this relationship seems evident, further research will be needed on several questions. Is the physical condition of the rehabilitated Class II C equivalent to that of the Class II C failures? What is the contribution of length of illness or length of time without work to the degree of family stability as rated by the caseworker?

ABILITY TO COPE WITH PREVIOUS LIFE STRESS. That ability to cope with previous life stress is related to successful rehabilitation is made clear by the analysis shown in Table 40-11, which indicates a decrease in the num-

ber of successes in all three functional classifications as we move from those rated *good* to those rated *poor*.

Before discussing the experience at the Altro Workshops, it may be noted that 13 of 14 clients who were dissatisfied with their work experience at the Altro were either full or partial successes as far as rehabilitation was concerned. The satisfied clients did not do quite as well, 40 of 61 being rehabilitated, and those who were neither satisfied nor dissatisfied did poorest with 21 failures as compared to 14 fully and 10 partially rehabilitated. It is also noted that 17 of 21 self-referrals were either fully or partially rehabilitated.

Ending Hours at the Workshop

No patient whose rehabilitation was a failure ever reached seven hours of work daily at the Altro Workshops and only one patient in functional classification II C who was rehabilitated reached seven hours of work daily while at the Workshops. All five patients in functional classification II C who reached only six or six and one-half hours were failures. The occupation most frequently represented before and after training at Altro was garment operation and its associated trades. Twenty-seven clients were in these trades before admission to Altro. Nine of these continued in garment operating after leaving Altro and five were still at the Workshops at the time of the study. Eleven clients who were in other occupations prior to Altro went into garment operating after leaving Altro (Table 40-3). The second most common occupation prior to training at Altro was factory, electrical, mechanical and building industry. There were 24 in this category before training and 10 after training at Altro. Three of the original 24 continued in this type of occupation after discharge from the Workshops. In only two occupations was there a rise in the number of employees so classified after discharge from Altro. Employees in clerical occupations rose from 6 to 15 and in the service occupations the number increased from 7 to 9.

Whatever else may be the significance of

these findings, the wide range of occupations to which the rehabilitated patient goes points up, again, the fact that the Altro Workshop is serving not as much as a place for vocational retraining but as a means for removing fears and restoring self-confidence in work capacity. At the same time this observation raises the question of whether or not there is a need for a more diversified program of vocational training.

Summary and Conclusions

Our chief findings with respect to the factors associated with success or failure may be summarized as follows:

1. The major factor related to success or failure in rehabilitation of those served at the Altro Workshops appeared to be the functional classification. Thus 93 per cent or 26 of 28 of those in the best classification, 65 per cent or 51 of 79 of those in the middle class, and only 35 per cent or 8 of 23 of those in the poorest category were either full or partial successes.

2. Of the medical factors, length of illness appeared to be next most important in relation to success or failure. In the middle functional classification, 55 per cent of those ill more than three years were failures as contrasted with 20 per cent of those ill less than three years. The three full successes in the poorest functional classification all had been ill less than three years. The five partial successes and 12 of the 15 failures had been ill for three years or longer.

3. In comparison with functional classification and length of illness, such factors as sex, nativity, ethnic background, age on admission, and etiology did not appear to be related strongly to success or failure.

4. With respect to work history, the length of time without work before admission was the factor most clearly related to success or failure. When this was added to functional classification, it became an even more effective index for predicting successful rehabilitation. Thus, 7 of the 8 successes in Class III C were out of work less than a year before admission, 13 of 15 failures in Class

III C had been out of work for more than a year before admission. The information regarding work history was considered inadequate for the other two failures.

5. Factors such as the stability of work before coming to Altro and the source of income also appeared related to success or failure. Only 2 of 15 patients with irregular work histories were full successes. Seventy-two per cent or 13 of 18 of those whose source of income was the Department of Welfare were failures.

6. There also appeared to be a trend for better education to be associated with successful rehabilitation.

7. The marital status appeared related to success. The married and widowed persons tended to be more successful than those divorced or separated, and those who were single appeared to have an intermediate potential for success.

8. The more favorable the current family stability and early family background, the greater was the chance for success in rehabilitation.

9. The greater the ability to cope with illness and the greater the ability to cope with previous life stress, the greater the rehabilitation potential appeared to be.

In conclusion, the Altro Workshops appeared to be highly effective in rehabilitating most of those patients in functional classification I B and I C and the majority of those in functional classification II C. Although the Workshops were least effective for those in functional classification III C, even among these clients 36 per cent were fully or partially rehabilitated. This is believed to be an indication that under sheltered conditions some kind of productive working life may be continued by patients with severely limiting cardiac disease.

ANALYSIS AND FOLLOW-UP OF 58 CLIENTS SERVED AT THE ALTRO WORKSHOPS

In the winter of 1955 to 1956, an analysis was made of an additional group of 58 clients served from August 1953 to August

31, 1955. The follow-up for 46 of these patients, who had been discharged from the Workshops, was done by mail and telephone and was, therefore, less intensive than that for the original group of 144. Nevertheless, it was possible to determine the employment status after training at the Altro Workshops for most of these individuals and to relate this status to many of the same factors studied in the original group of 144 clients. The intent was to provide a further check on those factors which appeared to be related to success or failure in rehabilitation at the Altro Workshops.

General Description

Study of the 58 additional clients reveals that they were similar to the original 144, although there were some differences.

Sex, Nativity, and Ethnic Background

While 48 per cent of the clients in both the original group of 144 clients (Group I) and the second group of 58 patients (Group II) were male, foreign born, and white, among the remainder there were only 2 per cent of Puerto Rican origin in the first group and 15 per cent with this background in the second (Table 40-12).

TABLE 40-12 DISTRIBUTION BY SEX, NATIVITY, AND ETHNIC BACKGROUND

Sex, nativity, and ethnic background	Group I		Group II	
	Num-ber	Per-cent	Num-ber	Per-cent
All patients	144	100	58	100
Male, native born	54	37	15	27
Male, foreign born	72	50	31	53
Female, native born	11	8	6	10
Female, foreign born	8	5	6	10

Functional Classification on Admission

The number of clients in the II C functional classification increased somewhat in Group II, and they still form a majority of the patients admitted. The number of those in the *best* category (I B, I C) rose slightly and there was some decrease in those in the *poorest* classification (III C) (Table 40-13).

TABLE 40-13 CARDIAC PATIENTS IN THE TWO PERIODS BY FUNCTIONAL CLASSIFICATION

Functional classification	Group I		Group II	
	Number	Per cent	Number	Per cent
All patients	144	100	58	100
I B	23	17	14	25
II C	87	60	39	68
II D-III C	25	18	39	7
Not established	1	1		

Age on Admission

While most clients in both groups fell into the age range of 46 to 55 years, there was a shift towards an older age level so that those aged 56 years and over formed 23 per cent of Group II as contrasted with only 16 per cent of Group I (Table 40-14).

TABLE 40-14 AGE DISTRIBUTION OF PATIENTS ADMITTED IN TWO PERIODS

Age on admission to Altro	Group I		Group II	
	Number	Per cent	Number	Per cent
All patients	144	100	58	100
18-25	11	8	3	5
26-35	14	10	8	14
36-45	43	30	10	17
46-55	53	36	23	40
56-60	22	15	10	17
61-65	1	1	2	3
Over 65	—	—	2	3

Etiology

There was little change in the relative number of patients in each etiologic category.

TABLE 40-15 ETIOLOGIC DISTRIBUTION OF PATIENTS ADMITTED IN TWO PERIODS

Etiology	Group I		Group II	
	Num-ber	Per-cent	Num-ber	Per-cent
All patients	144	100	58	100
Rheumatic heart disease	43	30	21	36
Arteriosclerotic and hypertensive heart disease	98	68	36	62
Congenital heart disease	2	1	—	—
TB pericarditis	1	1	1	2

although a rise from 30 to 36 per cent in the percentage of rheumatics in Group II was observed (Table 40-15).

Factors Associated with Employment Status After Altro Discharge

The 58 clients were not rated on a rehabilitation level. Their employment status after training at Altro was established by means of the follow-up. While this is not strictly comparable to the rating on a rehabilitation level that was used with Group I, some comparison of findings for the two groups may be attempted since the employment status after discharge from Altro was an important element in establishing rehabilitation rating for Group I.

Comparisons were made in this group of those who were employed either regularly or for a brief period after discharge from Altro and those who were unemployed. The finer distinctions between success, partial success, and failure in rehabilitation were not made. Of the 58 clients in Group II, 12 were still at Altro and no information on work history after discharge was available for one. The follow-up revealed that 27 of the remaining 45 clients or 60 per cent were either employed or briefly employed after Altro. This may be compared with 65 per cent fully or partially rehabilitated in Group I.

Factors in each of the four areas of our code were examined.

Medical Factors

FUNCTIONAL CLASSIFICATION Functional classification did not differentiate the employed from the unemployed as sharply as it did the rehabilitated from the nonrehabilitated in Group I (Table 40-16). Thus, of the 15 in the *best* functional classification, two were still at Altro and there was no information for one. Eight of the remaining 12 or 67 per cent were employed, either regularly or for a brief period of time, after training at Altro. Of the 32 patients in the *middle* functional classification, seven were still at Altro. Fourteen of the remaining 25 or 56 per cent were employed or briefly employed after discharge from Altro. Of the 11 in the

poorest functional classification, three were still at Altro. Five of the remaining eight or 62 per cent were employed or briefly employed after Altro.

TABLE 40-16 FUNCTIONAL CLASSIFICATION IN RELATION TO EMPLOYMENT STATUS AFTER DISCHARGE

Functional classification	Number			Total
	Number employed	briefly employed	unemployed	
Best (I B-I C)	7	1	4	12
Middle (II C)	12	2	11	25
Poorest (III C-D)	3	2	3	8
Total	22	5	18	45

Of the 18 unemployed clients, 9 or 50 per cent had rheumatic heart disease, 2 in the *best* and 6 in the *middle* functional classification. Etiology may therefore be a factor of influence here, cutting across functional classification when we are dealing with but a few patients. If the nonrheumatic patients, that is, those with arteriosclerotic and arterioscle-

TABLE 40-17 FUNCTIONAL CLASSIFICATION AND ARTERIOSCLEROTIC AND ARTERIOSCLEROTIC AND HYPERTENSIVE HEART DISEASE IN RELATION TO EMPLOYMENT STATUS AFTER DISCHARGE

Arteriosclerotic and hypertensive				
Functional classification	Number			Total
	Number employed	briefly employed	unemployed	
Best	7	—	2	9
Middle	8	2	5	15
Poorest	1	—	2	3
Total	16	2	9	27

rotic and hypertensive heart disease, are considered separately, then the positive relationship of classification to employment does hold. Seventy-eight per cent of those in the *best*, 67 per cent of those in the *middle*, and only 33 per cent of those in the *poorest* functional classification were employed or briefly employed after discharge from Altro (Table 40-17). Psychosocial factors may be of influence here. For example, the four failures,

two rheumatic and two arteriosclerotic, in the *best* functional classification include one separated and one divorced patient. The relation of marital status and family cohesiveness to successful rehabilitation is again noteworthy.

The comparison of the rheumatic and the arteriosclerotic and arteriosclerotic and hypertensive etiological classifications is shown in Table 40-18. Only 9 of 18 or 50 per cent

that with the original 144 cases this factor actually differentiated more sharply between success and partial success than between success and failure.

TIME WITHOUT WORK. If not quite as clearly as with the 144 clients in Group I, a negative relationship was noted again between time without work before coming to Altro and successful employment after training at the Workshop. The percentage of

TABLE 40-18 ETIOLOGY IN RELATION TO EMPLOYMENT STATUS AFTER ALTRO DISCHARGE

Etiology	Employed		Briefly employed		Unemployed		Total
	Number	Per cent	Number	Per cent	Number	Per cent	
Rheumatic heart disease	6	33	3	17	9	50	18
Arteriosclerotic and arteriosclerotic and hypertensive heart disease	16	59	2	7	9	33	27
Total	22		5		18		45

of the former who were discharged from the Workshops were employed or briefly employed after discharge from the Altro Workshops, as contrasted with 18 of 27 or 67 per cent of the latter.

LENGTH OF ILLNESS AT TIME OF ADMISSION TO ALTRO. As with Group I, the length of illness at the time of admission to Altro seemed to be a factor differentiating those who were to become employed from those to be unemployed. Fifteen of 23 patients or 65 per cent who were ill for 0 to 2.9 years were employed after discharge as compared with 11 of 21 or 52 per cent of those whose illness had been 3 to 20 years in duration. The findings for this smaller group are not as clear cut as for the larger group of 144 patients but they do seem to show a definite trend.

Work History and Education

STABILITY OF WORK HISTORY BEFORE ONSET OF ILLNESS. Since few of the patients were employed regularly before admission to the Workshops, it is difficult to judge whether stability of work was a factor that differentiated those who were later employed from those who were unemployed. It was noted

those who were employed or briefly employed after discharge dropped from 64 per cent for those out of work for less than two years to 50 per cent for those who had not worked for two to three years and to 42 per cent for those unemployed for three years and over. Furthermore, 83 per cent or 10 of 12 of those unemployed for less than a year before coming to Altro were employed at the time of the follow-up study.

Psychosocial Indicators

MARITAL STATUS. In the second group studied there was again an indication that marital status is related to work adjustment. Three of four or 75 per cent who were either divorced or separated were unemployed after training at Altro. In contrast, 18 of 28 or 64 per cent of the married clients and 7 of 10 or 70 per cent of the single patients were employed after training. Forty per cent of the widowed clients in Group I were unemployed as compared with 67 per cent of the widowed persons in Group II. We are dealing with too few subjects to provide a definitive conclusion but the general trend suggests that work adjustment is related to marital status.

Experience at Altro Workshops

LENGTH OF STAY. As with the first group studied, there was much variation in length of stay at the Workshops. There seemed to be no optimal time with the exception that eight of ten or 80 per cent of those patients who stayed from 9 to 15 months were subsequently employed. No other training period showed such high employment. There was only one client, unemployed, who stayed less than one week. As already noted, all five persons in functional Class II C in Group I who stayed less than one week were failures.

The study of the additional 58 patients served to substantiate, in some measure, the belief that factors such as functional classification, length of illness at time of admission, time without work before admission, and marital status are variables that influence the chance of success or failure of rehabilitation. As regards the use of the sheltered workshop for different types of cardiac patients, the evidence is that it is a highly effective means of rehabilitating almost all patients in functional classification I B and I C. It is also effective in the majority of patients in functional category II C. However, this latter classification is one that requires further refinement before it can be used to determine the degree of potential for a return to the work-a-day world.

The Problem of Functional Classification II C

While the Altro Workshops have helped a number of patients in the functional classification II C back to work, it is least effective with these persons. Most such individuals may be able to continue a productive working life only under permanently sheltered conditions. Individuals in Class II C are often also among the aged population and present a problem for the gerontologist that goes beyond the present scope of Altro's functions.

As noted previously, the two classifications that we have termed *best* (I B and I C) and *poorest* (III C) are rather effective tools for determining the potential for success or failure. The *middle* classification (II C) does not make sharp differentiation possible. The analysis of the factors associated with success

or failure has indicated which determinants in addition to functional classification may be used to predict success for individual patients. Among others, these include length of illness at time of admission, length of time without work before admission, marital status, and current family stability. An attempt to use these various factors in one scale will be made in a following section. For the proper evaluation of these factors the skills of the psychiatrist, psychologist, social worker, and vocational counselor are needed, as well as those of the cardiologist. At the same time, cardiologists are faced with the fact that the functional classification II C needs refinement in terms of its basis in the physical findings with respect to the individual.

Therefore, an attempt was made to analyze the physical findings for the functional classification II C alone. This analysis is shown as being worthy of further investigation and possibly to serve as a beginning for refinement of this functional classification.

HEART SOUNDS. Abnormal first heart sounds, including a gallop or accentuation, etc., were apparently a deterrent to rehabilitation. We cannot draw any conclusions from our observations of second heart sounds.

BLOOD PRESSURE. The systolic blood pressure did not appear to influence rehabilitation. We cannot draw any conclusions as to the influence on rehabilitation of the diastolic blood pressure up to levels of 120 mm Hg. When the diastolic pressure was above that level the chances of rehabilitation were poor. Whether blocking agents or the Kempner regime may change or alter the chances of successful rehabilitation has not been tested by us.

HEART FAILURE. Dyspnea together with any other sign of congestive heart failure was a strong deterrent to rehabilitation. Dyspnea, per se, was only a mild deterrent as long as it responded to digitalis and a diuretic regime. Hepatic enlargement together with pulmonary congestion was a strong deterrent. Precordial pain related to exertion was a deterrent. Precordial pain unrelated to exertion seemed to favor rehabilitation if this pain was not due to other serious illness. Exer-

It is noted that both successes and failures fell almost throughout the entire range, indicating that the scale needs considerable refinement. However, no failure, either in the arteriosclerotic and hypertensive or rheumatic group, scored below 3. Considered separately, rheumatics who scored 5.9 or less showed a higher potential for rehabilitation. With a score of 6 or more their chances were markedly reduced. Thus, 86 per cent or 11 of 13 of those who were fully rehabilitated, 62 per cent or 5 of 8 of those subsequently partially rehabilitated, and none of those who failed at rehabilitation received a score of 5.9 or less.

The chances for rehabilitation of the arteriosclerotic and hypertensive group also fell off markedly for those scoring above 5.9, with only 13 per cent or 6 of 46 of the fully rehabilitated in contrast to 79 per cent or 23 of 29 of the failures scoring so high. Note, however, that 56 per cent or 9 of 16 of the partially rehabilitated fell in the range beyond 5.9. It must be stressed that this partially rehabilitated group did not work in industry. From purely an industrial standpoint the score of 5.9 is a realistic one for the Altro sample.

The latest status of the patients in Group I as established by a follow-up in November, 1955, revealed that of the 13 rheumatics rated as fully rehabilitated with a score of 5.9 or less, 12 or 92 per cent were employed and for one there was no information available. Of the two clients in the fully rehabilitated category who scored 6 and over, one was employed and one unemployed.

Of the 15 rheumatics rated as failures in rehabilitation with a score of 6 and over (no rheumatic failure scored 5.9 or less), four or 27 per cent were unemployed, seven or 47 per cent were deceased, and for three or 20 per cent there was no information available. One patient was a permanently sheltered case at the Altro Workshops.

The latest survey of those in the arteriosclerotic and hypertensive group showed that of the 40 patients rated as fully rehabilitated and who scored 5.9 or less, 28 or 70 per cent

were employed; three or eight per cent were unemployed; four or ten per cent were deceased; and for three or eight per cent there was no information available. One patient was a permanently sheltered case at the Altro Workshops. Of the six considered fully rehabilitated and who scored 6 or more, two or 33 per cent were employed; one or 17 per cent was unemployed; one or 17 per cent was deceased; and two or 33 per cent were permanently sheltered at the Altro Workshops.

The follow-up in November, 1955, of 29 arteriosclerotic and hypertensive patients who were considered failures in rehabilitation, revealed that of the six who scored 5.9 or less, one or 17 per cent was employed, four or 67 per cent were deceased; and no information was available for one. Of the 23 failures with a score of 6 or above, there were none who were employed, eight or 35 per cent were unemployed, 11 or 48 per cent had died, one was attending school, one was permanently sheltered at the Altro; no information was available for one.

As a final word, it may be said that no scale can take into account the dynamic interplay of factors which, in the end, make for successful rehabilitation in the case of one individual or for failure in a second. Furthermore, under permanently sheltered conditions a number of patients with high scores on the scale were able to continue productive lives. An example was the rheumatic patient with a score of 9 who continued as a superintendent for a number of years after leaving the Altro Workshops because he had the able assistance of his wife. Successful rehabilitation depends upon the knowledge and skills of different disciplines acting in coordination to help the disabled cardiac back to productive living and self-respect.

COMMUNITY NEED: A BRIEF ANALYSIS

Two general approaches were used to evaluate the need of community for the services of the Altro Workshops. First, a follow-up was made of 88 clients who were provided a major assessment but who were not served at

the Altro Workshops. A major assessment consists of a diagnostic physical, social, psychological, and vocational evaluation. In addition, an analysis was made of the pattern of referrals during the period from 1948 through 1954 inclusive. In conjunction with this, interviews were held with twenty-four physicians whose patients had either been served at the Altro Workshops or had been given a major assessment by the Altro staff.

Follow-Up of 88 Clients Provided Major Assessment but Not Served at the Altro Workshops

One way of looking at a community need is to ask what happens to clients who might have, but did not, come to the Altro Workshops. Were those who were considered for Altro services, but failed to come, able to make their own adjustment to the working community and, if they did, how long did it take for such an adjustment? The problem of when a transition experience in a sheltered workshop may prove a "regressive" experience for the personality is touched upon here. Personalities are in various states of integration and have different environmental needs. Thus, it may be that often a cardiac needs only reassurance by the cardiologist and case-work help in clarifying his problems in order to be able to return to work. Indeed, a workshop experience for such individuals may be contraindicated. To answer some of these questions, a follow-up by mail and telephone was attempted for the 151 clients who had been examined by the Altro cardiologist from February, 1948, through December 31, 1954.

Eighty-eight of the 151 were reached through this follow-up. Since follow-up was not by personal interview, and since 34 of the 88 were deceased at the time of follow-up, the information for these 88 patients is not as complete as for those served at the Altro.

Description of Eighty-Eight Provided Major Assessment

A brief description of this group will set the background for the findings with respect

to their status at time of the follow-up. The majority were male, native or foreign born, white. The largest single group, 42 per cent, were in the functional classification II C (Table 40-21).

TABLE 40-21 FUNCTIONAL CLASSIFICATION OF 88 CARDIAC PATIENTS PROVIDED MAJOR ASSESSMENT

<i>Functional classification</i>	<i>Number</i>	<i>Per cent</i>
All patients	88	100
I B	25	28
II C	43	49
III C-III D	13	15
Not established	7	8

The age groups of 36 to 45 years and 46 to 55 years predominated, with 25 per cent and 35 per cent of the patients falling into these respective categories. The majority of clients, 63 per cent, had arteriosclerotic and hypertensive heart disease (Table 40-22).

TABLE 40-22 ETIOLOGIC CLASSIFICATION OF 88 CARDIAC PATIENTS PROVIDED MAJOR ASSESSMENT

<i>Etiology</i>	<i>Number</i>	<i>Per cent</i>
All patients	88	100
Rheumatic heart disease	31	35
Arteriosclerotic and hypertensive heart disease	55*	63*
Luetic heart disease	1	1
Not established	1	1

* Includes six patients with hypertensive heart disease.

The clients came from many occupations, although there were only 10 per cent garment operators in this group as contrasted with 21 per cent among those who were served at the Workshops.

It may be speculated that of those who accepted training, garment operators were readier to try the Altro program and therefore more represented in the group who were served. Most of the clients, 62 or 70 per cent, were classified as having had regular full time work before the onset of their illnesses.

Results of Survey of the 88 Cardiac Patients Provided Major Assessment

We turn now to the results of the follow-up survey of the status of the 88 cardiac patients provided major assessment. It was found that, of the total group, 51 or 58 per cent either had full or brief employment after contact with Altro or had gone to school; 24 per cent had no employment after contact with Altro, and no information concerning employment was available for 18 per cent. However, at the time of the follow-up only 33 per cent of the total group were employed or attending school. On the other hand, there were 39 per cent deceased and 28 per cent were unemployed. A similar analysis for patients in Groups I and II who were served at the Altro Workshops shows that 45 per cent were employed or attending school, 21 per cent were deceased, 25 per cent were unemployed, and no follow-up information was available for 9 per cent. It should be added that seven of those considered employed were working under permanently sheltered conditions at the Workshops and that these percentages do not include eight clients in Group II who were still in the course of rehabilitation.

With respect to functional classification, it was found that in the 88 cardiacs who were furnished only a major assessment at the Altro Workshops, the results were similar to those in the group of patients who were served at the Workshops. Those who fell into the better functional classifications showed a higher percentage of regular or brief employment than those in the poorer functional categories of the patients in whom information was available regarding employment after their contact with Altro. It was found that 20 of 21 or 95 per cent of the *best*, 21 of 31 or 67 per cent of the *middle*, and 7 of 13 or 54 per cent of the *poorest* functional classifications were employed. There were seven patients for whom no functional classification was established; only one was employed at the time of the follow-up.

Among the questions that are suggested for further investigation are the following.

Would the patients in the Major Assessment group have had results comparable to those found for those who were served at Altro if they had gone on to training at the Workshops? Would there have been more employed? Would fewer of them have died? The answers to these questions will require a carefully controlled study that should include control and experimental groups. The present study attempts in no way to provide the answers to these questions. However, we would like to call attention to the large number of deceased among this group and to the fact that ten Major Assessment clients took at least a year to find employment after their contact with the Altro Workshops.

Deceased

In all classifications among this group there was a large number of deceased at time of follow-up, 34 or 39 per cent. This compares with 40 or 21 per cent deceased at time of follow-up among those in both Groups I and II who had been served at the Altro Workshops. This percentage is based upon a total of 194 clients and does not include the eight clients who were still in the process of rehabilitation.

A breakdown of the causes of death for both the group provided Major Assessment and those served at Altro discloses that in the former, 89 per cent of the deaths were due to cardiac or cardiovascular causes. Fifty-nine per cent were cardiac, 9 per cent were cerebrovascular, and 21 per cent were cardiac and/or cerebrovascular, 5+ per cent were noncardiac deaths, and in 5+ per cent the cause of death was not established.

In the group who were served at Altro, 70+ per cent of the deaths were due to cardiac disease, 7+ per cent were cerebrovascular in nature, 7+ per cent were post-operative cardiac deaths, and 15 per cent were noncardiac deaths.

It is of interest to ask whether a period at Altro would have helped to prolong the lives of those individuals who were in the group provided only Major Assessment, either by providing them with an occupation that would not have proved a strain on their

limited capacities or by providing a testing period that would have determined whether or not, and to what extent, they could work.

Time Without Work after Altro Contact

The value of a sheltered workshop may also be judged in terms of the time it takes those served at the shop to return to work as compared to the time needed by those not served. There is some indication that those who were not served at Altro took a longer period of time to find employment after contact with the Altro Workshops than did those who were served. Of the 28 clients in the group provided assessment alone, who found employment and for whom information as to length of time to find employment was available, ten took longer than a year and five of these ten took two years or more. Would so long a period have elapsed if these individuals had gone through rehabilitation at the Altro Workshops? According to our records, only one of the 144 patients in Group I who were employed took longer than two years to find employment after discharge. None of those in Group II who were employed, and for whom information was available, took more than a year to find employment. The analysis of the 88 clients provided major assessment but not served at the Altro Workshops suggests that the Workshops may help

to speed recovery and possibly to prolong life. These are questions that certainly should be investigated more intensively.

As with other types of disablement and illness, in the field of cardiac rehabilitation there is no one method of service that is right for all clients. Depending on the client, his status at time of application, and his needs different kinds of treatment and rehabilitation are necessary. For some patients, sheltered workshop experience is extremely valuable. For others, other methods of rehabilitation are to be preferred.

The next step for investigation would seem to be to develop the criteria for the different methods of rehabilitation to be used with different kinds of client. From such an investigation will come the broader view with which the function of a sheltered workshop in the rehabilitation of the cardiac disable will be more readily seen and the most appropriate use will be made of the facilities it offers.

BIBLIOGRAPHY

- 1 JEZER, A., and HOCHHAUSER, E. Rehabilitation of the cardiac, *Brit J Phys Med* 17 1954
- 2 JEZER, A., and BLACK, B. J. Workshop experience with the disabled cardiac, *Brit Phys Med* 17 8, 1954

CHAPTER 41 THE SELECTIVE PLACEMENT OF CARDIACS IN INDUSTRY*

Neill K. Weaver

For many years certain workers at the Esso Baton Rouge Refinery who developed partially disabling cardiac conditions have been permitted to continue gainful employment. During the past five years a formal program for the placement of handicapped employees has been in operation. This program utilizes the matching of the worker's capacities with the physical demands of the job to achieve proper placement.^{1,2} An understanding refinery management, which fully recognizes the importance of the worker, "the company's most valuable asset," has contributed greatly to the success of this program.³ Company policies of self insurance and liberal employee benefits have tended to lessen those legal and economic factors which may act as significant deterrents to cardiac rehabilitation. In addition, the placement program has enjoyed a high level of co-operation on the part of the plant employees and their union officials.

The practicing physicians in the community have come to accept the refinery medical department as a *classification center* for cardiovascular diseases, and refer their employee-patients to it for evaluation of ability to work. In this setting, the refinery physicians have been able to follow a practice of encouraging rehabilitation at work whenever such a course appeared to be in the best interest of the patient's total health and well-being. It is believed that this approach to the cardiac rehabilitation problem is in accord with the views expressed by Master & Dack,⁴ Kresky & Goldwater,⁵ Franco,^{6,7} Hellerstein and Goldston,⁸ Dennison,⁹ and others

The physician in industry is particularly alert to the early detection of cardiovascular disease through the periodic examination program. The average refinery employee has had four such health inventories during the past

* Analysis of Experience at Baton Rouge Refinery of the Esso Standard Oil Company.

five years, and workers with suspected or known disease may be examined much more frequently. Patients report their symptoms or illnesses to the medical department at any time. Workers observed in apparent distress on the job are sent to the medical department immediately by their supervisor. Patients presumed to have heart disease are referred for study and placement by their personal physicians, as mentioned previously.

Evaluation and Placement of Workers with Heart Disease

Once recognized, the cardiac worker is evaluated by appropriate clinical technique: a careful history, thorough physical examination, electrocardiogram, ballistocardiogram, X-ray study or fluoroscopy, laboratory tests, pulmonary function studies (ventilatory tests), etc. The first objective of this evaluation is to arrive at an accurate etiologic and anatomic diagnosis of heart disease. A second and more important objective is to make a valid appraisal of the patient's functional capacity. We consider repeated interviews and examinations of utmost importance. This is particularly true in resolving cases of *possible heart disease*.

The patient's functional ability is determined in relation to those ordinary physical capacities which make up our activities on and off the job: standing, walking, climbing, lifting, carrying, etc. A Physical Capacities Analysis (PCA) form has been devised, upon which the physician indicates the number of hours permitted for each of these activities during the work day. He is then able, using practical, meaningful terms, to advise the patient, his family, and supervisor regarding the activities permitted.

The various job assignments in the refinery have been subjected to an engineering analysis, based on the same capacities; this is known as the Physical Demands Analysis. A medical management team, knowing the patient's qualifications and capacities, considers demands or requirements of various jobs. Proper placement of the limited employee is carried out by matching his capacities with the demands of the job (Fig 41-1).

Certain general aspects of the placement procedure are worthy of comment. The welfare of the patient should always have first consideration. The job should always be a legitimate one, not a makeshift or token assignment where the worker will be *carried* by fellow employees. The worker should be qualified or able to qualify for the job in terms of knowledge, training, and experience. The final decision or announcement should be made by a supervisor. We have found this point particularly important in obtaining acceptance by all parties concerned: the patient, his immediate supervisor, his fellow-workers, and his union representative. This is not so if the decision is made by the patient himself, his own doctor, or the industrial physician. It is usually desirable for the older worker to return to the same job or to a type of work similar to that done prior to the onset of disability. In addition, a careful system for follow-up, both on the job and in the medical department, is essential to insure suitability of the assignment.

Analysis of Results of Program for the Selective Placement of Employees with Heart Disease

To evaluate the results of the cardiac rehabilitation program, an analysis was made of all employees on the active roles of the refinery during 1956 whom the company physicians considered limited by heart disease. It should be emphasized that this study is not concerned with employees in whom there exists merely some medical evidence of a cardiac abnormality. All cases included for analysis have symptoms or findings on examination that warrant restriction of physical activities. In the nomenclature of the Criteria Committee of the New York Heart Association, they would be classified in Functional Capacity Class II or III, and in Therapeutic Class B (a few cases), C, or D (majority of cases).

The study revealed 200 workers disabled by heart disease, representing 2.8 per cent of the refinery's 7,140 employees. During 1956, 19 new cases were added to the refinery's pool of disabled cardinals, and 13 left the

Clinical Background of Case: Mr. A. B., age 57 WM Brick Mason. Myocardial infarction June 15, 1955.
 Uncomplicated recovery. Angina on walking 3-4 blocks at brisk pace. No evidence of congestive failure. Resumed work October 3, 1955 as Mason Toolroom Attendant.
 Is asymptomatic at work.

PLAIN
 PHYSICAL DEMANDS ANALYSIS

Numbers Hours Requires (Except Visual)
 F Pk, & 20
 one Code for Irritants and Toxic Materials

Mason Dept.

JOB TITLE & LOCATION

Brick Mason

Mason Toolroom Att'd

STAND	WALK	TALK	CLIMBING		LEGS	ARMS	LIFTING (UPWARD) INCLINING PUSHING & PULLING SHOULDER STRETCHING	CARRYING (UPWARD) INCLINING PUSHING & PULLING WALKING	NECK BEND	NECK TILT	NECK TURN	NECK EXT.	NECK FLEX.	NECK TIME
			ONLY	2	ONLY	2	ONLY	2	ONLY	2	ONLY	2	ONLY	2
11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
7	6	5	5	5	5	5	5	5	5	5	5	5	5	5
4	4	4	4	4	4	4	4	4	4	4	4	4	4	4

PLAIN
 PHYSICAL CAPACITIES ANALYSIS

A.B.

12345 57 10/3/55

Remarks

- #12. Ability to run is limited; may require rest periods.
- #13, 14. Rest may be required after each flight of stairs.
- #28, 29, 31, 32. May require rest periods to interrupt periods of effort.
- #42 Work must be done at a slower pace at temperature above 92°F.
- #43. Work must be done at a slower pace at temperature below 40°F.
- #54. Overtime should be minimized.

FIG 41-1 Thorough physical capacities analysis of the patient combined with engineering analysis of job requirements.

group: 11 by way of retirement (one by regular—age 65—retirement, three by early voluntary retirement, and seven by disability retirement) and two by death.

Etiologic classification of the 200 employee-patients is shown in Table 41-1. One hun-

TABLE 41-1 ETIOLOGIC CLASSIFICATION

Etiology	Patients	
	Number	Per cent
Arteriosclerotic	150	75
Infarction	91	46
Angina	59	29
Hypertensive	30	15
Rheumatic	11	5
Luetic	4	2.4
Miscellaneous	5	2.6
Total	200	

dred and fifty had arteriosclerotic heart disease, 91 with proved infarctions and 59 with angina or coronary insufficiency. The hypertensives, 30 in number, made up the second largest group. Eleven cases were of rheumatic origin, four cases were due to luetic heart disease, and the remaining five were due to other causes. Two of these had calcific aortic stenosis, two had arrhythmias (one with paroxysmal supraventricular tachycardia and the other with Wolff-Parkinson-White syndrome with runs of tachycardia) with bouts of congestive failure, and the final case was one of congenital heart disease (presumably an interauricular septal defect).

Clinical Status of Cardiac Workers

The arteriosclerotic heart disease group deserves special attention, since it represents such a challenge to the physician and the employer. The finding that three fourths of all the cardiacs discussed are disabled by arteriosclerotic heart disease is not surprising, in view of the aging plant population—50 per cent of the refinery workers are over 43.8 years of age. The age at onset (age at first infarction, or age at which work restrictions were imposed in the other instances) is shown in Table 41-2. Ten of the group with arterio-

TABLE 41-2 AGE AT ONSET OF DISABILITY

AGE	DISEASE					
	Infarct (91 cases)	Angina (59 cases)	Hypertension (30 cases)	Rheumatic (11 cases)	Luetic (4 cases)	Other (5 cases)
30-39 yrs	11	4	0	1	0	0
				(2 < 30)		
40-49 yrs	23	22	7	4	0	3
50-59 yrs	54	28	20	4	4	2
60-64 yrs	3	5	3	0	0	0

sclerotic heart disease were diabetics, and 14 had hypertension or a past record of high blood pressure. One case of myocardial infarction occurred in a patient with essential hyperlipemia, and another in a patient with Buerger's disease. Less than one fourth of the cases of myocardial infarction with a clear-cut symptomatic onset occurred during working hours.

Symptoms encountered in the various etiologic groups are summarized in Table 41-3.

TABLE 41-3 SYMPTOMS OF HEART DISEASE

	Angina			Failure	
	Mild	Moderate	Severe	Dyspnea	Edema
ASHD (150 cases)	75	19	0	26	10
Infarction (91)	28	7	0	14	2
Angina (59)	47	12	0	12	8
Hypertensive H.D. (30 cases)	5	0	0	14	6
Rheumatic H.D. (11 cases)	1	0	0	4	4
Luetic H.D. (4 cases)	0	1	0	1	1
Other etiology (5 cases)					
Calcific aortic stenosis (2)	1	0	0	2	0
Arrhythmias (2)	0	0	0	2	2
Congenital (1)	0	0	0	0	0

Of the 91 employees with healed infarcts, 43 were essentially asymptomatic on the amount of physical activity they followed on and off the job. Fourteen complained of exertional dyspnea, and two had experienced episodes of frank right and left ventricular failure. Thirty-five noted episodes of chest pain compatible with angina pectoris, this was considered mild (infrequent) in 28, and moderately severe (attacks occurring at intervals of a few days to monthly) in seven cases. Patients with severe angina were not permitted to continue at work.

In the *angina* group, bouts of chest pain occurred at infrequent intervals in 47 and at frequent intervals (every few days to monthly) in 12. Fourteen patients had suffered one or more definite episodes of coronary insufficiency, and 12 had experienced myocardial failure (predominantly left-sided in four, and both right and left-sided in eight).

Fourteen of the patients with hypertensive heart disease had symptoms of congestive failure, and five had angina. Practically all the patients in this group had X-ray evidence of cardiac enlargement, and electrocardiographic changes approaching or compatible with left ventricular hypertrophy or *strain* patterns.

had experienced bouts of failure. The patient with congenital heart disease, a female clerk, was asymptomatic; however, she exhibited X-ray evidence of an engorged pulmonary conus and possible right-sided enlargement of the heart. The only other female included in this series was a Negro maid with hypertensive heart disease and failure.

Work Status of Cardiac Employees

Table 41-4 shows the distribution of cardiac patients among the major job classifications. Actually, we have been able to place them in all types of refinery jobs, from executive to laborer. Of the 16 patients with infarcts working as supervisors, 11 were in the fore-

TABLE 41-4 WORK CLASSIFICATION

	ASHD						Total	
	Infarction	Angina	Hypertension	Rheumatic	Luetic	Other	Number	Per cent
Supervisory	16	3	2	0	0	0	21	10
Technical	8	3	1	5	1	0	18	9
Clerical	8	4	0	1	0	1	14	7
Skilled	42	37	9	4	0	2	94	47
Unskilled	17	12	18	1	3	2	53	27
Total	91	59	30	11	4	5	200	100

Four of the patients with rheumatic heart disease had overt symptoms of failure. Nine of the 11 had murmurs of aortic as well as of mitral valvular involvement, and three had a fixed rhythm of auricular fibrillation.

The four patients with luetic heart disease had the typical murmur of *open* aortic insufficiency and radiologic evidence of cardiac enlargement, but only one had experienced symptoms of failure. All had positive serologic tests and a history of syphilis many years before the availability of penicillin or other modern methods of treatment for the condition.

The two patients with calcific aortic stenosis complained of dyspnea and weakness, and one had angina and claudication; both had evidence of left ventricular hypertrophy. Both patients with the paroxysmal arrhythmias

man category, five being at a higher executive level. At the other end of the work scale, over one fourth of the entire group of cardiacs is in the unskilled worker category (mechanical and process helpers, laborers, etc.), and nearly half is in the skilled worker category (comprised of craftsmen of the various mechanical trades, and operators and assistant operators on refinery units). That three fourths of our cardiacs are able to work safely and effectively in these categories is highly significant. This is, undoubtedly, in part a reflection of the relatively high degree of mechanization and of the good environmental control (presence of air-conditioned mechanical shops, control rooms, etc.) in the refinery. It is also the result of a placement program that recognizes an individual's *capabilities*, rather than merely stressing his *lim-*

nations We have found that approximately one third of our employees are able to continue in the previous assignment with little if any change, and over half are able to do the same job with some alteration of its required activities The remainder (less than 20 per cent) must be assigned to a new job.

The frequency of occurrence of cardiac workers is consistent throughout all job categories of the refinery male population (Table 41-5) Those differences which are present

TABLE 41-5 INCIDENCE OF DISABLING HEART DISEASE IN REFINERY MALE EMPLOYEES

Group	ASHD Per cent	M HD Per cent
Supervisory-administrative (765 employees)	2.5	2.8
Technical-clerical (1,047 employees)	2.2	2.9
All salaried (1,812 employees)	2.3	2.9
Skilled (3,059 employees)	2.6	3.0
Unskilled (1,990 employees)	1.6	2.7
All wage earners (5,049 employees)	2.1	2.9
All male workers (6,861 employees)	2.2	2.9

would seem to be readily explained on the basis of age, the skilled wage earners (2.6 per cent with arteriosclerotic heart disease) being a significantly older group than the unskilled workers (1.6 per cent with arteriosclerotic heart disease) Consequently, we feel that no particular work group is vulnerable to heart disease.

Cardiac workers are effective on the job. It is noteworthy that the partially disabled employee can be properly placed with no resultant loss in effectiveness or productivity. Only four of the 200 cardiac workers were on the supervisors' list of *substandard* employees during 1956. In one of these, a machinist assigned to maintenance work on a unit, the physical disability resulting from inadequately controlled congestive failure was the cause of ineffectiveness; he received a disability retirement in early 1957. In two cases

a cardiac neurosis was a major contributory cause of *substandard* work status. The fourth case appears to be purely one of poor motivation for work.

The amount of sickness absenteeism attributable to heart disease for the whole group was not high. Excluding absences that were the direct result of myocardial infarction (see below) or disability retirement, the average for each employee was only one time on *sick report* for a total of seven calendar days each year. No figures are available for a suitable control group, but this represents an absenteeism experience roughly equal to that of the average refinery worker for all medical causes. An excellent study of this subject has recently been presented by Thorpe.¹⁰

TABLE 41-6 MONTHS SPENT ON SICK REPORT BEFORE RESUMING WORK
(91 Patients with 96 myocardial infarctions)

Number of months	Number of patients
None	5
1 Month	12
2 Months	16
3 Months	28
4 Months	11
5 Months	12
6 Months	6
7+ Months	6

Table 41-6 shows the number of months spent on sick report by patients following a myocardial infarction. That 17 were off the job a month or less (no time lost in five cases) may seem surprising. In many of such cases, the *infarct* was clinically *silent*, and the diagnosis made in retrospect. In other uncomplicated cases with evidence of very minor myocardial damage, the patient may be permitted to resume part-time work of a very light nature (clerical work, for example) after six or eight weeks of convalescence. We feel that the majority of patients with uncomplicated infarctions can return to light work after three or four months. It has been our experience that patients with prolonged invalidism are often unable to return to

work, owing to a disabling cardiac neurosis or symptoms of severe angina or failure.

The period worked by the partially disabled cardiac employees is shown in Table 41-7. The average number of years worked

TABLE 41-7 YEARS AT WORK WITH CARDIAC DISABILITY

	Number of patients		
	1½-2 Yrs	2-5 Yrs	5+ Yrs
ASHD	32	71	35
Infarction	16	39	29
			(max 11 years)
Angina	16	32	7
			(max 12 years)
Other etiology	4	34	9
			(max 8 years)

by each employee who has been successfully placed in an assignment six months or longer is as follows: myocardial infarction group, 4.3 years; angina group, 3.3 years (3.9 years for the whole group with arteriosclerotic heart disease); and 3.4 years for the other etiologic classes. The entire cardiac group has a total work experience of 702 man-years of productive employment.

Summary

Experience with the selective placement of industrial workers with heart disease is reviewed. Methods by which employees with cardiac disability may be identified in an industrial population are described. By means of careful clinical evaluation, their capacities for work may be determined. Availability of an engineering analysis of various jobs, expressed in the same capacities, permits proper placement by matching the patient's capacities with the demands of the job.

The clinical status of 200 workers with impaired cardiac function who have received job assignments in a large refinery in such manner is described in detail. Their work status is reviewed with respect to job classification, job effectiveness, sickness absenteeism, and the number of years worked with cardiac disability.

The data presented indicate that industrial workers who develop cardiac disability may continue to work effectively through proper placement.

BIBLIOGRAPHY

1. HANVAN, BERT. *Physical Capacities and Job Placement*. Stockholm, Nordisk Rotogravyr, 1951.
2. HANSEN, H., and WEAVER, N. K. Arteriosclerotic hearts at work, *J Louisiana M Soc* 107:63, 1955.
3. *Statement on Medical Policy*. Esso Standard Oil Company, 15 West 51st Street, New York, May 8, 1956.
4. MASTER, A. M., and DACK, S. Rehabilitation following acute coronary artery occlusion, *JAMA* 155:828, 1940.
5. KRESKY, B., and GOLDWATER, L. J. Occupational potentialities of cardiac patients, *Am Heart J* 27:623, 1944.
6. FRANCO, S. C. Cardiovascular disease in industry, role of degenerative disease, *Indust Med* 20:308, 1951.
7. FRANCO, S. C. The cardiac can work, *Indust Med* 23:315, 1954.
8. HELLERSTEIN, H. K., and GOLDSTON, E. Rehabilitation of patients with heart disease, *Postgrad. Med* 15:265, 1954.
9. DENNISON, A. D., JR. Evaluation of the cardiac in industry, *Indust. Med* 23:164, 1954.
10. THORPE, J. J. The optimum time for return to work following various major cardiovascular disabilities, *Indust Med* 25:329, 1956.

CHAPTER 42 A STUDY OF EMPLOYEES WHO DEVELOPED MYOCARDIAL INFARCTION*

C. A. D'Alonzo and Sidney Pell

On January 1, 1956, the Medical Division of the Du Pont Company initiated a company-wide, long-term follow-up study of employees who developed an acute myocardial infarct. A case is included in the project if there is no prior history of a myocardial infarction and if the diagnosis of the current infarction is confirmed by clinical evidence with typical electrocardiographic findings, or by post-mortem reports. The observed population comprises approximately 90,000 employees, located throughout the country, ranging in age from 17 to 64 years. The present study is an analysis of cases that were reported during 1956.

Sources and Nature of Data

Cases are made known routinely by records maintained for two of the company's employee benefit plans: one that provides life

insurance and another that furnishes funds for medical expenses in the event of non-occupational disability whose duration exceeds seven consecutive days. To be eligible for health insurance, an employee must have at least six months of continuous service. The plan is voluntary, and requires a nominal monthly premium payment. The participation rate among those eligible is about 96 per cent. Life insurance is available to all employees who have at least one year of continuous service. The entire cost of the plan is paid by the company, and participation is universal among those eligible. Since employee participation in these insurance plans is very high, one might expect very few, if any, cases to go unreported because of non-participation. The population base used to compute incidence rates is adjusted to include only those employees who are covered by one or both insurance plans.

When a case is made known to us, the medical records of the affected employee are

* A study of 209 employees of the E. I. du Pont de Nemours and Company who developed myocardial infarction during 1956.

obtained from his company physician. These records contain clinical and laboratory findings of the annual physical examinations provided by the company for all employees, reports of visits to the plant dispensary, dates and causes of sickness absenteeism, correspondence with the family physician and hospitals, and other information relevant to the health of the employee. The following items of information are extracted from these records for use in the present study: sex, year of birth, occupation, blood type, height, and 10-year record of body weights, blood pressure readings, and pulse rates. Note is also made of findings obtained from electrocardiograms and chest x-rays. The records are reviewed by us for confirmation of the diagnosis of myocardial infarction.

Objective of the Present Study

It is hoped that, eventually, the follow-up of cases will provide data to investigate the effect of a myocardial infarction on the working life of the afflicted employee and to identify factors that affect survivorship rates. At the present stage of the project, however, the study objectives are limited to an inquiry into the following questions:

1. What is the magnitude of the problem as it exists in our company?
2. How do incidence rates vary according to age, sex, occupation, and season of the year?
3. To what extent are the chances of recovery related to age, excess body weight, hypertension, and a history of tachycardia?

Incidence

During the year, 1956, the cause of disability or death was described on 252 insurance claims as one of the following: "coronary occlusion," "coronary attack," "heart attack," "myocardial infarction," or "coronary thrombosis." After a review of the medical records, 43 cases were excluded from the study, 13 because of insufficient evidence of a myocardial infarction and 30 because of a history of myocardial infarction prior to 1956. The remaining 209 cases comprise the subject matter of this paper.

Geographical Distribution

The subjects were employed in 49 company installations in 18 states. About 70 per cent (146 cases) were reported from locations situated in five of these states. New Jersey (46), Delaware (41), Virginia (22), New York (19), and Tennessee (18). Approximately 90 per cent of the subjects resided in the Middle Atlantic and Southeastern states.

Age and Sex Variation

Of the 209 cases, 207 were males and two were females. The incidence rates in the age range 25 to 64 years were 2.9 cases per 1,000 male employees and 0.2 cases per 1,000 female employees. Both females were between 60 and 64 years. The wide difference between the sexes in incidence of the disease and the absence of any female cases in the premenopausal years demonstrate well-known phenomena. Age-specific incidence rates are shown in Table 42-1 and Figure 42-1. Below

TABLE 42-1 INCIDENCE OF INITIAL MYOCARDIAL INFARCTION BY AGE AND SEX, 1956

Age	Male		Female	
	Number	Cases per 1000 employees	Number	Cases per 1000 employees
25-34	3	0.1	0	0
35-44	43	1.8	0	0
45-54	87	5.7	0	0
55-64	74	11.3	2	2.0
Total	207	2.9	2	0.2

the age of 35, there were three cases among the male employees. The youngest of these was 26 years old. In the age group 35 to 44, the incidence among the male employees was 1.8 per 1,000. The rate increases to 5.7 in the age group 45 to 54, and to 11.3 between the ages of 55 and 64.

Seasonal Variation

The number of attacks that occurred during each month of the year is shown in Table 42-2. The largest number occurred during

the month of July, when 30 were reported, while February, with nine cases, had the lowest incidence. It is very unlikely that the observed monthly variation could have arisen as a result of chance alone ($P < .01$)

The pattern of seasonal variation may be seen graphically in Figure 42-2. It is clear that the incidence was highest during the

summer months, July through September. During this period of time, 76 cases were reported. The winter months, January through March, with 36 cases, had the lowest seasonal incidence. These data suggest that the risk of developing a myocardial infarction is about twice as great during the summer as it is during the winter.

The seasonal differences observed in this study are contrary to those reported by several other investigators. In their series of 240 cases, Billings and his associates¹ found that the incidence was highest during the winter and lowest during the summer. Bean² noted a low incidence during the summer, but no substantial differences among the other seasons. Data presented by Mintz and Katz³ show minimal monthly variation throughout the year, except for a slight increase during late fall and early winter. McVay,⁴ on the other hand, reported a seasonal low during the winter. In his study of 47 cases among Air Force personnel, only four occurred from December through February.

There does not appear to be any explanation for the inconsistencies in these findings. They may have arisen because of differences in the selection of cases. The seasonal pattern

TABLE 42-2. INCIDENCE OF INITIAL MYOCARDIAL INFARCTION BY MONTH, 1956

	Number of cases	
	Monthly	Quarterly
January	13	
February	9	
March	14	36
April	14	
May	17	
June	13	44
July	30	
August	23	
September	23	76
October	17	
November	21	
December	15	53
Total	209	209

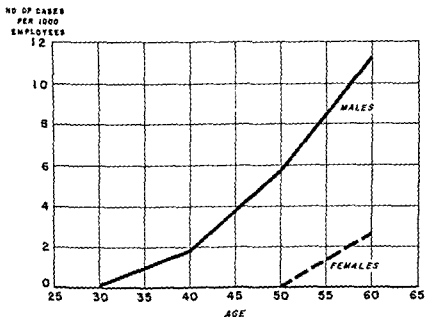


FIG 42-1. Age-specific incidence rates of initial myocardial infarction, by sex, 1956

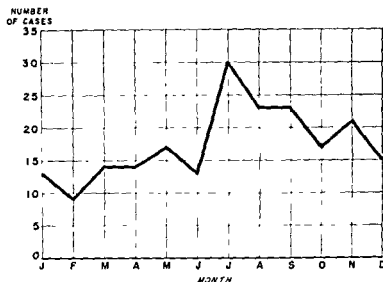


FIG. 42-2. Incidence of initial myocardial infarction during each month of 1956

we observe during 1957 should help clarify this problem

Factors Associated with Recovery

For purposes of this study, the subjects have been divided into two categories with respect to survival after the attack. An employee is classified as a "recovered" case if he survived 30 days or longer, while an employee who died within 30 days of the attack is designated a "case fatality." Of the 209 cases reported during 1956, 139 recovered and the remaining 70 died within 30 days. This is a case fatality rate of 33.5 per cent.

In this section of the paper, the data on the male cases are analyzed to determine whether the chances of surviving 30 days or longer are associated with the following factors: age, hypertension, excess body weight, and a history of tachycardia.

Age

The case fatality rate in each ten-year age group is shown in Table 42-3. Although the rates increase in each successive age category, differences among these rates can be attributed to sampling variation. It is possible, however, that as cases are added to the study the present pattern may persist. If that does happen, a positive association between age and case fatality will be demonstrated statistically. Increasing mortality with age has been reported in the literature^{3,5,6}

TABLE 42-3 AGE-SPECIFIC CASE FATALITY RATES AMONG 207 MALE CASES OF INITIAL MYOCARDIAL INFARCTION, 1956

Age	Total	Recovered	Died*	Case fatality rate (per cent)
25-34	3	3	0	0
35-44	43	32	11	25.6
45-54	87	57	30	34.5
55-64	74	45	29	39.2
Total	207	137	70	33.8

* Died within 30 days of the onset of the acute attack

Hypertension

To classify the cases as hypertensive or not hypertensive, reference was made to the blood pressure readings that were taken during the past ten years at the time of the employee's periodic physical examination. An employee is designated a hypertensive if his medical records show two successive annual readings of 150 mm Hg or more, systolic, or 94 mm Hg or more, diastolic.

According to these criteria, 54 cases in the study were classified as hypertensive. The case fatality rate in this group was 37.0 per cent. Among the 153 nonhypertensives, the rate was 32.7 per cent. The difference between the two rates is not significant.

Several other investigators^{1,2,3,7,8} have also

STUDY OF EMPLOYEES WHO DEVELOPED MYOCARDIAL INFARCTION

379

TABLE 42-4 CASE FATALITY RATES AMONG MALE CASES OF INITIAL MYOCARDIAL INFARCTION WITH AND WITHOUT HISTORY OF HYPERTENSION, * 1956

	Number of cases			Case fatality rate (per cent)
	Total	Recovered	Died†	
Hypertensive	54	34	20	37.0
Not hypertensive	153	103	50	32.7
Total	207	137	70	33.8

* Two successive annual readings of 150 mm Hg or more, systolic, or 94 mm Hg or more, diastolic
† Died within 30 days of the onset of the acute attack

reported that a pre-existing hypertension bears no relationship to prognosis following an acute myocardial infarction

Excess Body Weight

To study the effect of excess body weight on chances of surviving a coronary attack, the subjects were classified as overweight if their body weight, as measured within a year prior to the attack, exceeded their ideal weight by 20 per cent or more. For purposes of this study, the ideal weight for a specified height is the weight midway between the allowable extremes for a person of medium frame, as recommended in the height and weight tables of the Metropolitan Life Insurance Company. Because of practical difficulties, it was not feasible to obtain the employees' somatotypes or specific gravities, or

other information that would be useful in determining degree of obesity

Seventy-two of the 207 male cases were classified as overweight. The case fatality rate in this group was 36.1 per cent, and in the group classified as not overweight, the rate was 32.6 per cent. The difference of 3.5 per cent can be attributed to chance variation

TABLE 42-5 CASE FATALITY RATES AMONG MALE CASES OF INITIAL MYOCARDIAL INFARCTION, WITH AND WITHOUT EXCESS BODY WEIGHT, * 1955

	Number of cases			Case fatality rate (per cent)
	Total	Recovered	Died†	
Overweight	72	46	26	36.1
Not overweight	135	91	44	32.6
Total	207	137	70	33.8

* Twenty per cent or more over ideal weight
† Died within 30 days of the onset of the acute attack

History of Tachycardia

The data were analyzed further to determine whether a history of tachycardia bore any relation to case fatality rates. Reference was made to the records of pulse rates taken at periodic physical examinations during the ten years preceding the attack. If any two pulse rates were 90 or more, the employee was classified as having a history of tachycardia. The 34 male cases with such a history had a case fatality rate of 29.4 per cent. The

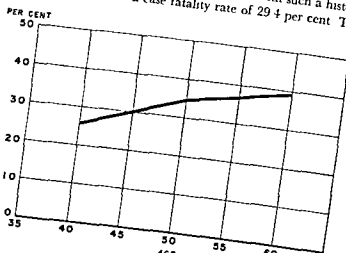


FIG 42-3 Age-specific case fatality rates among male cases of initial myocardial infarction, 1956

rate among cases without a history of tachycardia was 34.7 per cent. The difference between the two rates is not significant.

TABLE 42-6. CASE FATALITY RATES AMONG MALE CASES OF INITIAL MYOCARDIAL INFARCTION, WITH AND WITHOUT HISTORY OF TACHYCARDIA, * 1956

	Number of cases			Case fatality rate (per cent)
	Total	Recovered	Died†	
History of tachycardia	34	24	10	29.4
No history of tachycardia	173	113	60	34.7
Total	207	137	70	33.8

* Two or more pulse rate readings of 90 or over taken at periodic physical examinations during the ten years preceding the attack.

† Died within 30 days of the onset of the acute attack.

Thus, there is no evidence in the present series of data that the chances of recovery are associated with age, hypertension, excess body weight, or a history of tachycardia.

Study of Preinfarction Electrocardiogram

Eighty per cent of our cases had one or more electrocardiograms prior to their attack. The number and kind of abnormalities noted

TABLE 42-7. NUMBER OF CASES WITH CERTAIN ABNORMALITIES NOTED ON ELECTROCARDIOGRAMS TAKEN PRIOR TO ATTACK

Abnormality	Number	Per cent
Bundle branch block		
Right	2	1.2
Mixed or incomplete	2	1.2
Heart block	3	1.8
ST-T abnormalities	6	3.6
T wave changes	11	6.5
Premature auricular contractions	2	1.2
Premature ventricular contractions	7	4.2
One or more abnormalities	27	16.1
No abnormalities	141	83.9
Total who were given one or more EKG's prior to attack	168	100.0
Number with no EKG's prior to attack	41	
Total number of cases	209	

prior to the attack are found in Table 42-7. Of particular interest is the fact that, of the 168 subjects who were given electrocardiograms prior to the attack, approximately 84 per cent showed no evidence of cardiac abnormalities. These data illustrate the well-known inadequacy of the electrocardiogram as a means of predicting a coronary attack or detecting the presence of atherosclerosis.

Location of the Infarct

The location of the infarct was known in 121 (57.9 per cent) of our subjects. The distribution of the location, as shown in Table 42-8, indicates a preponderance of posterior

TABLE 42-8. LOCATION OF INFARCT

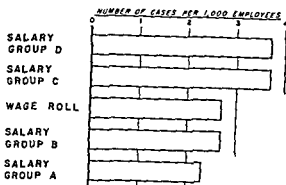
	Number	Per cent
Posterior	62	51.2
Anterior	34	28.1
Antero-septal	14	11.6
Posterior-lateral	7	5.8
Antero-lateral	2	1.7
Anterior and posterior	1	0.8
Lateral intramural	1	0.8
Total—Location known	121	100.0
Location unknown	88	
Total number of cases	209	

infarcts. The ratio of posterior to anterior, specified as such, is 1.82 to 1 (62 to 34). Other studies, however, report a majority located in the anterior wall.^{1,6,8} This difference may be due in part to our lack of information in 88 of our cases.

Occupational Variation in Incidence of Myocardial Infarction

The company employees are divided into two broad categories for payroll purposes. One is the wage roll and the other, the salary roll. The wage roll group consists chiefly of laborers and skilled and semiskilled production workers. The occupations of the salary roll employees are mostly sedentary in nature. They include clerical, professional, administrative, and supervisory personnel. For purposes of this study, the salaried employees have been subdivided into four categories, each one descriptive of how the occupations within the category are rated with respect to

FIG 42-4 Age-adjusted incidence rates of initial myocardial infarction by occupational classification (Male employees, 25-64 years; 1956)



Employees On The Salary Roll Are Classified According To Degree Of Job Responsibility On A Scale Extending From A, The Highest Level, Through D

their relative degree of responsibility. The categories are designated A, B, C, and D. Group A includes the top-level managers and executives; D, clerical workers with a minimum of responsibility; and B and C, employees whose job responsibility lies between the two extremes.

Incidence rates of myocardial infarction in each of the five occupational groups described above are shown in Table 42-9. Because of

TABLE 42-9 AGE-ADJUSTED INCIDENCE RATES OF INITIAL MYOCARDIAL INFARCTION BY OCCUPATIONAL CLASSIFICATION* (Male employees, 25-64 years, 1956)

Occupational classification	Number of cases	Age-adjusted rate
Salary roll		
Group A	5	2.3
Group B	29	2.7
Group C	36	3.7
Group D	16	3.7
Total salary roll	86	3.2
Total wage roll	121	2.7

* Employees on the salary roll are classified according to degree of job responsibility on a scale extending from A, the highest level, through D.

the large differences in the age distribution of these groups, it was necessary to adjust the rates to remove the effect of the age differences.*

Variation in incidence of myocardial in-

farction among the five occupational categories is not any greater than one might expect to find as a result of chance alone. It is interesting to note, however, that the age-adjusted rates, as shown graphically in Figure 42-4, were highest in Groups C and D, the salaried employees with the least job responsibility, and lowest in Group A. These findings are not consistent with the hypothesis that executives and other high-ranking personnel are most susceptible to attacks of coronary thrombosis.

A similar conclusion was reached by Stamler in his analysis of mortality due to cardiovascular-renal diseases in Chicago, Illinois.* The determination of any real differences among the occupational groups described in this study must await the accumulation of additional data.

Summary

Two hundred and nine employees of the Du Pont Company in 49 company installations located in 18 states developed an acute myocardial infarction during 1956. In the age range 25 to 64 years, the incidence was 2.9 cases per 1,000 male employees and 0.2 cases per 1,000 female employees. Only two of the subjects were females, and both were between 60 and 64 years of age. The youngest male in the series was 26 years old. Incidence of attacks was observed to increase sharply with age.

Further analysis of data revealed the following:

1. Incidence was highest during the summer and lowest during the winter.

* The rates were adjusted by the indirect method. The standard rates used in the calculations were the age-specific attack rates for the company as a whole.

2. Approximately one third of the subjects died within 30 days of the acute attack

3. The chances of dying within 30 days of the acute attack appear to increase with age, but our experience in the present study is not large enough to exclude the possibility that age variation in case fatality is due solely to chance.

4. There is no evidence that the immediate prognosis is affected by overweight, a pre-existing hypertension, or a history of tachycardia

5. No significant differences in incidence of myocardial infarction were found among occupational groups classified according to degree of job responsibility

BIBLIOGRAPHY

- 1 BILLINGS, F. T., KALSTONE, B. M., SPENCER, J. L., BALL, C. O. T., and MENEELY, G. R.: Prognosis of acute myocardial infarction, *Am J Med* 7 356, 1949
- 2 BEAN, W. B.: Infarction of the heart, morphological and clinical appraisal of 300 cases,

predisposing and precipitating conditions, *Am Heart J* 14 684, 1937

3. MINTZ, S. S., and KATZ, L. N.: Recent myocardial infarction; analysis of 572 cases, *Arch Int. Med.* 80.205, 1947.
4. McVAY, L. V., JR.: Myocardial infarction in young men, *Postgrad. Med.* 20 506, 1956
5. MASTER, A. M., DACK, S., and JAFFE, H. L.: Age, sex, and hypertension in myocardial infarction due to coronary occlusion, *Arch Int Med.* 64 767, 1939
6. ROSENBAUM, F. F., and LEVINE, S. A.: Prognostic values of various clinical and electrocardiographic features of acute myocardial infarction, immediate prognosis, *Arch Int Med* 68-913, 1941
7. MASTER, A. M., JAFFE, H. L., DACK, S., and SILVER, N.: Course of blood pressure before, during, and after coronary occlusion, *Am Heart J* 26-92, 1943
8. FISHER, R. L., and ZUCKERMAN, M.: Coronary thrombosis, *JAMA* 131 385, 1946.
9. STAMLER, J.: Epidemiologic investigation of group mortality trends in Chicago due to arteriosclerotic and hypertensive cardiovascular disease, *Circulation* 12.497, 1955.

CHAPTER 43 EXPERIENCE IN SELECTIVE PLACEMENT AND FOLLOW-UP ON CARDIACS*

William E. R. Greer

It has been suggested that under proper job placement and satisfactory mental and emotional states cardinals in industry should exhibit a good report insofar as work efficiency, absenteeism, accidents, and need for special considerations are concerned. Moreover, there is increasing evidence that the etiology of arteriosclerotic heart disease rests more with poor health habits and sedentary living than with hard work, overexercise, or occupational stress.

Working capacity evaluation of the cardiac worker must rest on careful clinical judgment and mental capabilities of the employee in relation to the requirements of the job to which he is to be assigned or re-assigned.

It is imperative that the cardiac worker be acquainted with his physical status and that appropriate health counseling for main-

tenance of optimal health from both physical and emotional standpoints be accomplished.

In the pilot study of a plant of 2,600 workers that we have undertaken, a number of observations would appear to be of interest. The incidence of cardiac disease in the plant population was about 8 per cent of the employees examined (Table 43-1). It

TABLE 43-1 INCIDENCE OF CARDIOVASCULAR DISEASE AMONG 2,600 GILLETTE EMPLOYEES

	Total		Per cent
	Males	Females	
Essential hypertension	63	37	100
Arteriosclerotic heart disease	43	2	45
Rheumatic heart disease	11	13	24
Hypertensive cardiovascular disease	13	5	18
Abnormal electrocardiograms	10	2	12
Congenital heart disease	1	2	3
	141	61	
	(70%)	(30%)	

* Analysis of 202 workers with cardiovascular disease employed at the Gillette Safety Razor Company.

should be noted that there are about twice as many males as there are females in the group surveyed. The incidence of cardiovascular disease in any group carefully examined averages about 10 to 12 per cent.

It is significant that 65 per cent of the group are in the age group 40 to 60 years (Table 43-2). This is the time of greatest

TABLE 43-2 AGE GROUP INCIDENCE OF CARDIOVASCULAR DISEASE

	Under 30 yrs	30-39 yrs	40-49 yrs	50-59 yrs	60-69 yrs
Essential hypertension	3	17	31	36	13
Arteriosclerotic heart disease	0	2	7	26	11
Rheumatic heart disease	7	5	7	6	0
Hypertensive cardiovascular disease	1	0	4	8	5
Abnormal electrocardiogram	0	1	6	5	0
Congenital heart disease	1	2	0	0	0
Percentage	6	13	25	40	14

responsibility and a time when earning capacities are most important. The 19 per cent of employees with cardiovascular disease under 40 years of age may represent a significant future disability or compensation problem.

Since we have not examined 100 per cent of all the employees, the total significance of the figures in Table 43-3 is not forthcoming.

TABLE 43-3 INCIDENCE OF CARDIOVASCULAR DISEASE BY OCCUPATIONAL CATEGORY

	Man- agement	Clerical	Skilled	Un- skilled
Essential hypertension	18	10	48	24
Arteriosclerotic heart disease	11	1	16	15
Rheumatic heart disease	1	8	5	10
Hypertensive cardiovascular disease	1	4	7	6
Abnormal electrocardiogram	5	2	3	2
Congenital heart disease	0	2	1	0
Percentage	19%	13%	40%	29%

However, there are approximately 100 members of the management group, and, as indicated, 38 per cent of them have evidence

of cardiovascular disease. These are the leaders or ones with greatest responsibility. There has been no restriction of advancement in position because of evidence of cardiovascular disease. There has been no evidence of aggravation of the disease process.

Arteriosclerotic Heart Disease

Arteriosclerotic heart disease has occupied a major position in discussions of the cardiovascular worker. In this pilot study we have noted 4 employees in this category. There was significant family history in 12 (26 per cent) of this group. Three of these employees had diabetes mellitus. There was one instance of hypercholesterolemia in a 36-year-old salesman who has had two myocardial infarctions but who continues to perform a vigorous schedule. Twenty-four (53 per

TABLE 43-4 INCIDENCE OF MYOCARDIAL INFARCTIONS

Number of cases in arteriosclerotic group, 27 (60%)	
Age at the time of first myocardial infarction	
Age in years	Number of employees
30-39	2
40-49	9
50-59	12
60-69	4
Years of work since initial myocardial infarction	
1-4 years	13 employees
5-9 years	10 employees
10-15 years	4 employees
Employees with multiple myocardial infarctions, 5	
Change in employee status since 1955	
Deceased	4
Cleared	1 (not cardiac reasons)
Retired	2 (not cardiac reasons)

cent) of this group exhibit angina pectoris to some or marked degree. Ten (22 per cent) of the arteriosclerotic heart disease group are under treatment for chronic congestive heart failure. Concerning body weight, it is of interest that 11 per cent weighed 110 to 140 pounds; 35 per cent weighed 140 to 160 pounds; 22 per cent weighed 160 to 180 pounds; 18 per cent weighed 180 to 200 pounds, and 13 per cent weighed over 200 pounds on initial observations.

atic Heart Disease

re have apparently been few studies
fate of the employee with rheumatic
disease. It is almost routine in most
ries in the area to reject any pre-em-
ent applicant who has a heart mur-
Industry is worried about the possible
ive disability problem if rheumatics
ized. In the group under study there
twenty-four known cases of rheumatic
disease (Table 43-5). In the employees

TABLE 43-5 RHEUMATIC HEART DISEASE

Types of valvular disease in 2600 employees	Number of cases
A stenosis	3
I stenosis and insufficiency	3
I insufficiency	14
I and aortic stenosis and insufficiency	3
I stenosis	1
Total	24

t rheumatic heart diseases, five exhibited
ence of congestive heart failure. Eleven
loyees showed significant cardiac en-
ement. Four employees had embolic
nomena. Eight of these individuals were
iving digitals and other cardiac medica-
s. One of these employees had successful
ral valve surgery. Since 1955, one of
se employees has died and two have been
ared but not for cardiac reasons. Over a
g period of study this group may repre-
it a potentially greater industrial risk. It
of interest to note the almost equal distri-
tion of cases in this group by age decade
(Table 43-2), employees with evidence of
ibolic phenomenon are the worst risks in
is group.

Occupational Accidents or Injuries

There have been few studies of the car-
ac employee with regard to liability or
ceptibility to occupational accidents or
juries. In this study cardiacs seemed to
ave more accidents than the plant average
lthough none were significant. There have
een no cardiac death claims or claims for

TABLE 43-6 INCIDENCE OF ACCIDENTS IN 202 CARDIAC EMPLOYEES

	Number of employees	Major accidents	Minor accidents
Essential hypertension	100	3	14
Arteriosclerotic heart disease	45	0	9
Rheumatic heart disease	24	2	3
Hypertensive cardiovascular disease	18	0	1
Abnormal electrocardiogram	12	0	2
Congenital heart disease	3	0	0
Accidents		5	29

Incidence of major accidents, 2.6 per cent (Over 2 weeks' absence).

Incidence of minor accidents, 14.0 per cent (No time lost).

Incidence of major accidents in 100 unselected non-cardiac employees, 1 per cent.

Incidence of minor accidents in 100 unselected non-cardiac employees, 2 per cent.

aggravation of existing heart diseases (Table 43-6).

Absenteeism in Cardiac Employees

The breakdown of absenteeism in the plant population for 1956 in regard to employees with specific cardiovascular disease as compared to the so-called average employee is shown in Table 43-7. These statis-

TABLE 43-7 ABSENTEEISM

	Average days lost per year per employee
Average employee	11.3
Male	8.98
Female	15.3
Essential hypertension	11.3
Arteriosclerotic heart disease	13.3
Rheumatic heart disease	13.6
Hypertensive cardiovascular disease	10.6
Abnormal electrocardiogram	6.6
Congenital heart disease	3.3

tics in a small continuing study certainly represent a most favorable comparison.

If one pursues reasons for even these low absenteeism rates for cardiac employees, other interesting observations are noted. There were 259 days' absenteeism in the essential hypertension group for reasons un-

TABLE 43-8 WORK PERFORMANCE RATING OF CARDIAC EMPLOYEES

	Premium	Regular	Unsatisfactory
Essential hypertension	30	39	3
Arteriosclerotic heart disease	8	23	2
Rheumatic heart disease	7	9	3
Hypertensive cardiovascular disease	7	9	0
Abnormal electrocardiogram	3	4	1
Congenital heart disease	1	1	0
Percentage	36	55	7

related to hypertension. About 50 per cent of the employees with hypertension had less than three days absenteeism for the whole year. Twenty-one of the employees accounted for 785 days out of 1,077 days' total absenteeism in the group. In the group with rheumatic heart disease there were eight employees who accounted for 240 out of 314 days total absenteeism. There were 87 days lost for noncardiac reasons. Nine employees (40 per cent) had less than three days' absenteeism. In the arteriosclerotic group twelve employees accounted for 462 out of 600 days' total absenteeism. There were 152 days lost for noncardiac reasons. Twenty-one (46 per cent) of this group had less than three days' total absenteeism. In the group with hypertensive vascular disease five employees accounted for 148 out of 212 days' total absenteeism. There were 78 days lost for noncardiac reasons. Thirteen (72 per cent) of these employees had less than three days' total absenteeism.

In this pilot study to date cardiacs would appear to exhibit a good record where absenteeism is concerned. In our studies of total long-term absenteeism diseases of the musculoskeletal system, respiratory system, and female reproductive system represent a much greater cause of time lost from work than do diseases of the cardiovascular system.

Work Performance Rating

An important factor in hiring or rehiring the cardiac worker is a consideration of his work performance. The average plant population shows 20 per cent premium workers and 50 per cent regular workers. The work performance of the cardiac workers in this study compares most favorably with the average healthy employee (Table 43-8), 36 per cent of the cardiac employees were premium and 55 per cent were regular in work performance. There were seven per cent in the base performance group and one per cent unsatisfactory. It is of interest that 31 per cent of the employees with essential

hypertension show premium performance when the average for the plant population is 20 per cent. This is in keeping with the "hypertensive personality" of aggressive, conscientious, hard-driving characteristics.

Instances where special considerations had to be given were rare. These individuals have not represented a safety hazard, have produced at a profit, and usually have excellent morale effects on fellow workers. There has been no objective evidence of aggravation of the disease process.

For industry as a whole studies should be made on a broad basis as to the relative hazards, the accident proneness, the compensation risks, the work performance, and the average absenteeism of the worker with cardiac disability. These factors will vary with certain industries. Care and planning can salvage much valuable productivity that is now being wasted. Industrial populations present ideal situations for observations and research in certain features of cardiovascular disease.

Summary

A pilot study of cardiovascular disease in a plant of 2,600 workers is presented.

About 8 per cent of the employees examined exhibited evidence of cardiovascular disease.

It would appear that the cardiac employees in this group showed an excellent record insofar as work performance, absenteeism, hazards involved, and need for special considerations are concerned.

CHAPTER 44 MYOCARDIAL INFARCTION IN GARMENT WORKERS

CHARACTERISTICS AND RELATIONSHIP TO TIME LOST FROM WORK

*Leo Price**

CHARACTERISTICS

Introduction

Myocardial infarction that occurs among workers deserves the interest and discussion it is now receiving. Since this disease has a definite economic and social impact upon our society, there is obvious value in studying the influence of coronary heart disease upon industrial workers.

In the past, myocardial infarctions with survival usually implied serious loss of health, prolonged temporary or permanent disability, and an outlook of a limited life span. They also implied distressingly high medical expenses and long periods of confining illness. Moreover, they were associated with financial

losses both to the workers and to the employer. For the former there were serious immediate losses of wages, and family hardships because of unemployment. For the latter there were production losses owing to absences of older, skilled and dependable workers in whom the employer had heavy capital investments.

Published reports concerning acute and subacute cases of myocardial infarction generally deal with the hospital patient. This study of garment workers with coronary heart disease who have experienced myocardial infarctions is based upon medical supervision of the ambulant patient who visits our clinic at regular intervals.

Included in this study are some workers who were employed when the infarction was discovered, it was found that some continued at their employment regardless of medical advice to rest during the acute or subacute stages of coronary disease activity. It was

* The appreciation of the Author is extended to Mr. Sidney Sameth, Chief Statistician of the Union Health Center, for the statistical problems solved in the course of this Survey, and to Dr. Harold Marcus and Dr. Alexander Oscharoff for their participation in the study.

found that 85 per cent of the patients surveyed in this study went back to work after recovery from the attack.

Background of Industry's Medical Care Program

The patients under consideration are employed in the ladies' garment industry, which has 200,000 workers in the New York City area. These workers are provided with prepaid ambulatory medical care at the Union Health Center. During an average day, the Center serves between 1,400 and 2,000 patients, who are primarily in the older age groups. The Center's medical service emphasizes preventive medicine and utilizes frequent laboratory examinations, as well as miniature chest x-rays.

In 1955, 46,000 70 mm fluorographic chest films and more than 15,000 electrocardiograms were taken. While about 80 per cent of the workers in this industry are women, only 60 per cent of the patients at the Center are female. Most of the male patients are in the older age groups.

Heart disease has always been an important entity that has faced the physicians at the Center, as evidenced by the large number of claims for sickness insurance for temporary and partial disability owing to heart disease and the large number of cardiac cases treated in the clinic. In addition, the examinations of retirement applicants showed this category of disease to be a leading cause of premature retirement at age 60 and over. Furthermore, the greatest and most frequent cause of death in the industry has been from heart disease, averaging 46 per cent of the 1,300 annual deaths for the last five years.

Material Presented in This Study

In 1955, 15,112 electrocardiograms were performed upon 13,184 individual patients, 11,613 persons had one electrocardiogram done, 1,295 had two electrocardiograms in the course of the year, 215 patients had 3 electrocardiograms done that year; 60 patients had 4, 5, or 6 electrocardiograms done, and 1 patient had 7 electrocardiographic tracings in the course of the year.

Among the patients who had electrocardio-

graphic tracings in 1955, 732 showed myocardial infarctions; 1,703 had myocardial damage, and 767 had other abnormalities indicating heart disease. There were 3, patients with abnormal tracings and 9, patients with normal readings.

The 732 cases diagnosed as showing electrocardiographic pattern of myocardial infarction were carefully reviewed by Dr. Marcel Goldenberg, our Chief Electrocardiographer.

A Myocardial Infarction Register was initiated with these 732 cases and every subsequent case of myocardial infarction discovered among the patients at the Center has been added to this record. A total of 8 of these patients have died at this writing.

Age and Sex of Patients with Myocardial Infarctions

Table 44-1, analyzing the age and sex of 732 infarction patients, shows that myocardial infarctions occurred predominantly in the fifth and sixth decades of life in both men and women. Among the men, 51 per cent occurred in the 60-69 age group and 30 per cent in men between 50 and 59 years of age. Similarly, in the women the largest number of infarcts occurred in their sixties and the next largest group of infarctions occurred in women in their fifties.

The ratio of infarctions in all these 732 patients was 5.2 in men to 1 in women. In patients more than 50 years of age the ratio of infarction was 5.8 in men to 1 in women.

Many of the patients examined had one or more of the complications usually met with in older age, chronically ill persons. The most frequent complication was hypertension, followed by diabetes, obesity, and anxiety state.

Study of 204 Cases of Myocardial Infarction

In this group of 204 patients whose myocardial infarction was discovered for the first time in 1955, it was found that 73 cases, or 35.8 per cent, were verified as having an acute myocardial infarction that had occurred within eight weeks of the taking of the electrocardiogram. This is the proportion in which ambulant acute cases of myocardial in-

TABLE 44-1 A AGE AND SEX OF PATIENTS WITH MYOCARDIAL INFARCTIONS, B AGE AND SEX OF PATIENTS ATTENDING THE HEALTH CENTER

Age groups	A Infarction patients registered in 1955				B Patients attending center during one month in 1955			
	Male		Female		Male		Female	
	Number	Per cent	Number	Per cent	Number	Per cent	Number	Per cent
70 & over	81	13.2	2	1.7	1,077	19.6	279	3.0
60-69	314	51.0	54	46.2	2,341	42.5	2,137	23.2
50-59	188	30.6	45	38.5	1,119	20.3	3,392	36.7
40-49	27	4.4	14	12.0	479	8.7	2,058	22.3
Under 40	5	0.8	2	1.6	492	8.9	1,365	14.8
Total	615	100.0	117	100.0	5,508	100.0	9,231	100.0
Per cent of male and female	x	84.0	x	16.0	x	37.4	x	62.6

infarction were encountered in this health center (Table 44-2).

The balance of the 131 cases showed on electrocardiographic diagnosis either subacute or chronic myocardial infarctions that were seen for the first time in these patients, although most of them had visited the Center

TABLE 44-2 MYOCARDIAL INFARCTIONS IN AMBULANT PATIENTS FIRST RECORDED IN 1955

	Number of patients	Per cent
Acute myocardial infarctions	73	55.8
All other myocardial infarctions	131	64.2
Total myocardial infarctions	204	100.0

previously and had had electrocardiograms which did not show an infarction pattern.

From this group, and from the rest of the 732 infarction cases, registered in 1955, the material for the disability study was secured.

Electrocardiographic Procedure

Although the 12 lead electrocardiograms are being done in selected cases, the data here presented are from the following 7 leads:

The 3 limb leads, the unipolar lead AVF, and the following chest leads V₂, V₃, V₅.

Because the 7 lead electrocardiogram is the basis of the data, it is possible that some infarctions were overlooked. However, be-

cause of the electrocardiographic department's conservative and realistic view, and because of the great volume of work, it is felt that additional routine leads may be omitted for the present.

Characteristics of Myocardial Disease Encountered at Center

Many of the cases encountered at the Union Health Center gave the impression that they were "benign" in character, when one considers the lack of symptoms and signs, the work history, and the survival period. Sometimes the symptoms were so mild and few as to be ignored or not recognized by the patient. Sometimes the impression gained after persistent questioning was that pain and other symptoms of coronary disease were altogether absent. Apparently we meet a type of myocardial infarction that either involves a small area or else the infarction develops slowly. Moreover, because of the age of the patient, he is likely to have good collateral circulation.

In some instances the infarctions occurred in patients who had been under long supervision for arteriosclerosis, coronary insufficiency, myocardial damage, or angina. Nevertheless, when an acute infarction in the ambulant patient was demonstrated electrocardiographically and clinically substantiated by some new or unusual symptoms such as a new type of pain, cold perspiration, vomiting, or "weakness," it has been a problem at times to convince the patient that he had a criti-

cal illness, that he should discontinue work immediately and accept hospitalization.

Therefore, this type of infarction seen among older workers differs from the typical, serious, readily recognizable case of myocardial infarction in which the symptoms vary from severe shock with pain and breathlessness, to the moderately severe forms of coronary attacks conventionally described. While the conventional type was also encountered, many of the cases in this series did not have an immediately serious prognosis, some had relatively short periods of disability, and 27 per cent had no disability whatsoever.

The assumption is that in these ambulant cases of acute myocardial infarction a lesion developed any time up to eight weeks prior to the performance of the electrocardiogram at the Center. This time period is based upon the observation that even the larger lesions will have healed within two months.¹

All other cases, which include subacute and chronic myocardial infarction, are grouped together. Herein are included cases who had normal electrocardiograms during attendance at the Center until an electrocardiographic tracing taken during a subsequent clinical visit showed an infarction pattern that had no evidence of recency. It also includes a variety of cases with no previous electrocardiograms in their records, and whose first electrocardiogram showed a pattern of past infarction.

Many of the cases were of the subacute type and while under observation continued to show changes in the pattern of the electrocardiograms taken periodically. Regardless of the progression or regression of the lesion, the very presence of instability implies extension or contraction of a myocardial lesion, and underlines the serious nature of the condition and the need of careful supervision, particularly when the patient insists upon working.

Sometimes it is utterly impossible to verify clinically the date of the occurrence of the actual attack. This eludes the physician regardless of the care, persistence, and time taken to investigate the history and to record the symptoms. The inability to fix the clinical date of the incident is because the patient

was not aware of the attack and sought medical attention. Either he attended clinic irregularly or there were long intervals between visits. Whenever the patient had experienced symptoms verifying an infarction the actual clinical date was noted. In cases of acute infarction, with no symptoms, the electrocardiographic date was recorded as the date of infarction. In subacute or chronic cases who reported no symptoms, the date of the infarction was arbitrarily fixed at a midpoint between the date of the electrocardiogram showing an infarction pattern and the date of the previous visit at which an electrocardiogram showed other than an infarction pattern, providing it was less than two years.

Some of the patients denied having felt any pain whatever, in spite of persistent questioning. Some recognized nothing unusual in the mild to severe pain they experienced prior to or during an attack. Often no history could be obtained of change in pain location, radiation, or intensity, or whether the attack was accompanied by symptoms other than those previously experienced. Some patients were completely "unaware" of experiencing a heart attack, although we have electrocardiographic evidence either of an acute myocardial infarction or of an infarction that had occurred sometime during the extended interval between visits to our institution. Some of the patients refused to accept medical advice to stop work, and thus lost no time from their usual employment.

It would appear that a fair number of ambulant patients experience no pain whatever during the myocardial infarction. Some may have symptoms such as nausea, vomiting, vertigo, or an attack of weakness or fainting. But these identifying symptoms are comparatively uncommon in the painless infarction incidents.

Absence of pain has been reported in the literature, but absence of pain is not the rule. Its frequency of occurrence in our ambulatory clinic is higher than the 5 to 10 per cent reported.²

We have focused our attention upon pain primarily, although not exclusively, as an

identifying symptom in myocardial infarction. We recognize the numerous varieties of pain, ranging from mild distress to the excruciating, crushing pain that brings fear of impending death. We have considered the location of the pain, its radiation, and the time factor. It is well recognized that some persons are highly insensitive to pain. It is, however, only the indefinite, mildly distressing types of pain that might be overlooked by the patients studied. The crushing, severe types of pain with pressure on the anterior chest, with or without radiation to the arms, would not result in a questionable or negative history.

It is not necessary to describe the many other symptoms and signs that are characteristic of acute myocardial infarction. But repeated physical examinations, with all diagnostic facilities used, have given assurance of the accuracy of the diagnosis, in spite of the absence of fever, blood pressure changes, increased sedimentation rates, or evidence of heart failure.

The primary purpose of discussing pain, although other symptoms are explored and considered, is that it has been most common and useful in identifying which cases were unaware of the occurrence of an infarction identifiable on electrocardiogram. In studying disability, we have not included cases showing auricular fibrillation, heart block, and other cardiac irregularities.

This study, therefore, emphasizes not only that numerous acute myocardial infarctions occur among ambulant patients, but also that some such patients are "unaware" of the occurrence of a heart attack. The symptoms and signs develop in persons who are either insensitive to mild pain or are accustomed to more severe pain and are not prone to seek medical care for "minor" complaints.

It also includes so-called silent cases of coronary disease that may be entirely free from symptoms and signs. Among garment workers attending an ambulant health center, many such cases continue work through the period of a heart attack and lose little or no time from work. The discovery of the condition was only possible through frequent

and/or routine electrocardiograms of workers visiting the Center for check-up examinations or for medical care for complaints other than heart disease.

RELATIONSHIP TO TIME LOST FROM WORK

Disability

For the specific purpose of estimating loss of time from work because of disability, we made a survey of active patients with a record of a single infarction who were included in the 1955 Infarction Register. Data were gathered on these patients who attended the Center for medical care, not necessarily for cardiac complaints.

In the limited time available to perform this Study (October, 1956, to February, 1957), 219 patients and their records were reexamined. Among the group were 33 who had experienced the infarction after retirement from the industry. They were eliminated from the tabulations.

The study and re-examination of these patients was necessarily performed during the day hours of the health center services. For this reason active working patients, who seek medical service in the evening clinics after working hours, were not reached in sufficient proportion.

The selection, therefore, was not considered wholly representative of a cross section of the industrial group, since it contained many workers who seek medical care more frequently than the average clinic patient, as well as some workers who are the first to quit at the close of the work season and the last to seek a return to their machines as the new season commences.

This current Study is being continued to include a larger and more representative number of patients who worked in the industry after infarction.

For our purposes the "disability period" was considered time lost from work because of illness. Due to the seasonal nature of the industry, the disability period in some instances may have been lengthened by the slack season in which no work was available.

A total of 202 of the 216 patients were

TABLE 44-3 PERIOD OF DISABILITY CAUSED BY INFARCTION
(216 cases: 157 cases with disability, 59 with no disability)

Disability in weeks	Total patients	Patients hospitalized	Total weeks disability	Per cent of patients surveyed	Per cent of patients hospitalized	Cumulative percentages	
						Total patients (157)	Hosp. patients (92)
None	59*	0	0	x	x	x	x
Up to 3 weeks	12	6	26	7.6	50.0	7.6	6.5
4-6 weeks	20	9	101	12.7	45.0	20.3	16.3
7-9 weeks	23	9	181	14.7	39.1	35.0	26.1
10-13 weeks	32	22	341	20.4	68.8	55.4	50.0
14-26 weeks	50	31	915	31.9	62.0	87.3	83.7
27-52 weeks	20	15	770	12.7	75.0	100.0	100.0
Total	157	92	2,334	100.0	58.6	—	—

* Cases that had no disability are not included in total or percentages

Jewish, 10 were Italian, two Irish, one Spanish and one Negro. There were 191 men and 25 women in the group. Among them 2,334 weeks of working time were lost owing to the infarction. Fifty-nine of these patients, 27.3 per cent, had lost no time from work at all.

Table 44-3 shows that of patients who had disability about one third of the cases (35 per cent) lost nine weeks or less of employment owing to the infarction. Of the 55 patients disabled nine weeks or less, 24, or 43 per cent, were hospitalized; 102 patients were disabled 10 weeks or more, of whom 68, or 66 per cent, were hospitalized.

This high hospitalization rate for patients disabled for a relatively short period may be indicative of the tendency of these workers to take advantage of the better medical care available in recent years. In this industry the prepaid hospital insurance program now aids hospitalization, and generally better economic conditions may be overcoming the reluctance of these workers to accept hospital care.

It was found that the 92 patients hospitalized spent 4,311 days in hospital, an average of 47 days per patient.

The average disability period for the patients who were disabled was 14.9 weeks (see Table 44-4). The disability among the surveyed group of 216 cases with myocardial infarction was 10.8 weeks.

TABLE 44-4 COMPARISON OF DISABILITY PERIODS
TOTAL 216 MYOCARDIAL INFARCTION CASES COM-
PARED WITH 157 CASES WHO HAD DISABILITY

	Total 216 cases	157 cases with disability
Per cent patients under 9 weeks disability	52.8	35.0
Per cent patients under 13 weeks disability	67.6	55.4
Per cent of patients hospitalized	42.5	58.6
Average weeks disability	10.8 wks	14.9 wks
Weeks of disability in percentages		
No disability	27.3	0
Up to 3 weeks	5.6	7.6
4-6 weeks	9.3	12.7
7-9 weeks	10.6	14.7
10-13 weeks	14.8	20.4
14-26 weeks	23.1	31.9
27-52 weeks	9.3	12.7
	100.0%	100.0%

Survival

Table 44-5 reveals that 57.9 per cent of the patients studied, 125 persons, were 60 years of age or more at the time of the infarction. Thirty-two of this group of workers over 60 had infarctions from 6 to 10 years earlier. Three patients over 70 years old and 49 between 60 and 69 years of age had the infarction within the last two years.

Of the 216 patients studied we have found that 112 are working now and 104 are retired, all of the latter over the age of 65. Thirty-three retired immediately after the attack, while 71 went back to work after the infarction and eventually retired.

In the past decade, with the development of an industrial pension program in this industry to supplement federal Social Security payments, garment workers have become more prone to accept the idea of retirement, although at this time better wages and shorter hours make their jobs more attractive

TABLE 44-3 AGE OF WORKERS AT THE TIME OF INFARCTION AND ELAPSED PERIOD

Number of patients	Per cent of total	Age at time of the infarction				Years elapsed since infarction (as of 1955)
		Under 40	50-59	60-69	Over 70	
87	40.2	8	27	49	3	1-2
52	24.1	2	12	35	3	3-5
61	28.2	4	25	30	2	6-10
11	5.1	4	4	1	0	11-15
1	0.5	1	0	0	0	16-20
4	1.9	1	3	0	0	21-30
216	—	20	71	117	8	
Percent	100.0	9.3	32.9	54.1	3.7	

In this industry some older workers are, because of their skills, welcomed back by their employers after their illnesses. However, all who wish to return to work can do so primarily because their union contract guarantees job security.

Disability claims do not always reflect the degree of severity of the heart conditions of the claimants. The reasons for the length of time lost from employment are complex, varied, and subject to many influences. Working conditions, wage levels, job security, insurance coverage, savings, marital status, domestic environment and satisfactions, the age and the sex of the patient all seem to share in determining the time lost from work because of illness.

To a most important degree, disability is dependent upon the attitudes and the medical service of the attending physician. Recent

advances in medical treatment, the availability of more medical care to workers, publicity and health education concerning the seriousness of heart disease also seem to affect the time taken by cardiacs for convalescence.

Therefore, in reviewing the sick benefit program of the last ten years in the garment industry, we find that when maximum disability benefits covered 10 weeks and payments were small, the average time lost from work was short. When maximum benefit periods increased to 13 weeks and weekly payments were advanced, the average disability period lengthened. Now that disability payments have further increased and the period has been extended to 20 weeks, the time lost from work in all likelihood will be further prolonged.

Back in 1950, when the motivation to return to work was great because of limited disability benefits and low annual income owing to short seasonal work, disability periods in general were short. At that time the maximum indemnity for disability was \$26 a week for 13 weeks a year (having been raised from \$15 weekly for 10 weeks) and the average disability period for coronary disease, including hypertensive and arteriosclerotic heart disease, was about 6 weeks.

Because in this industry the worker has job security through his union contract, if he is able to produce he may return to his former job regardless of the time lost from disability. On the other hand, in spite of the present high industrial employment records, workers in other industries without job security are not assured of a return to their former employment after a heart attack. Moreover it is with difficulty that they secure a job elsewhere.

The rising costs of Workmen's Compensation Insurance for heart disease disability has caused considerable alarm in industry, and there have been several reports released and recommendations made for the solution of this problem.

In the State of Utah a plan has been adopted by common agreement under which all claims for disability owing to heart disease are referred to a panel of cardiologists recom-

mended by the medical societies and appointed by the industrial commissioner. The panel determines the causal relationship between the heart attack and the claimant's employment and advises the industrial commissioner concerning the compensability of the disability. It would be premature to pass judgment on whether such a plan could be wholly acceptable. However, continued trial is merited.

It would be well to study the intent and purpose of the Workmen's Compensation Law and to review the recent trend whereby a large section of industry has accepted responsibility for the health and welfare of employees, as well as for the costs of hospitalization, surgery, and medical care for the families of its workers.

Industry already in part accepts the concept that it must provide financial protection to employees with long records of service who age and deteriorate in harness and succumb to degenerative diseases with ensuing disabilities. Apparently there is a parallel to the depreciation that results from long wear and tear on machinery, which is added to the costs of operation. This growing realization of industry's responsibility for the health and

welfare of employees covers temporary partial disabilities resulting from both occupational and nonoccupational illnesses.

Further protection for the older employee is now provided by the federal Social Security program, which has extended the pension program to cover workers permanently disabled after age 50. Recently the addition of total and permanent disability clauses to industrial pension programs has helped spread the costs of heart disease in the older worker who may be unable to return to employment.

The integration of the many voluntary governmental programs for the assistance of disabled workers might help to still the alarm and remove the basis for the controversy concerning the problem of whether the cause of disability from heart disease is occupational or nonoccupational.

BIBLIOGRAPHY

- 1 MALLORY, G. K., WHITE, P. D., and SALCEDO, J. Speed of healing of myocardial infarction, study of pathologic anatomy in 7 cases, *Am. Heart J.* 18: 647, 1939.
- 2 POLLARD, H. M., and HARVILL, T. H. Painless myocardial infarction, *Am. J. M. Sc.* 199: 628, 1940.

CHAPTER 45 EMPLOYMENT PROBLEMS IN PERSONS WITH CARDIOVASCULAR DISEASE

E. A. Irvin

The employment problems in connection with the individual with heart disease are somewhat different from the problems confronting us in regard to many other diseases. However, the general principles of job placement apply in this condition as in any other physical condition of the employee. Proper job placement is the keynote to the efficient utilization of the cardiovascular patient. We must remember that a *successfully placed* handicapped worker ceases to be handicapped from the standpoint of earnings or productivity.

The problems involved in this question might be considered from the standpoint of the (1) patient, (2) private physician, (3) industrial physician, (4) employer, and (5) labor union. The private physician, the specialist, the employer, the industrial physician and the labor unions must be more conscious of the Golden Rule in relation to the individual who has heart disease. We must exercise good judgment in the placement of

these individuals at work and show an attitude of fairness to the patient and the employer in recommendations that are based on our medical knowledge and experience. We should place the individual on a job for which he is physically qualified without putting an extra burden on his cardiovascular system. Recognition must be given to the humanitarian and economic factors from the viewpoint of the patient—he wants work—he must work—and he will work if given an opportunity.

The private physician usually understands the patient's physical condition but often lacks a true understanding of the patient's problem in regard to his work. This may be due to many factors. Frequently the patient desires to work and in many instances economic status is such that he will mirror the effort required on his job in order to the approval of his physician for his return to work. Many physicians have very little knowledge of the various jobs within

tory and are in a poor position to approve the patient for his regular work. Much of this could be avoided if the private physician would contact the industrial physician in regard to the patient's work and also inform the industrial physician relative to the patient's condition.

It is embarrassing when there is a conflict of opinion, as, for example, when the private physician approves the patient for regular work only to have the patient disapproved for return to his regular work when examined by the plant physician. The patient naturally questions the wisdom of one of the decisions. This could be avoided easily by closer co-operation between the two physicians. Another factor that must be considered in relation to the employment of the cardiac is his means of transportation to and from work. Does he have to stand on a corner waiting for a bus, make several transfers and then have a long walk at the end of the line? Does he have to walk upstairs to his locker room? These all sound like simple questions but they are frequently very important to the patient.

The industrial physician plays a very important part in any job placement program. However, it is frequently difficult for the industrial physician to evaluate the patient's physical capacity for work when he has not had the opportunity to follow the patient in the course of his illness. In such cases, consultation between the private physician and the industrial physician proves helpful to the patient. We believe that all advice and recommendations to a patient or employee returning to work should be in writing to the industrial physician. If this is not practical, a telephone call is helpful.

A sound examination plan is the basis for a good health maintenance program. The physical examination is essential to a job placement program and selective job placement is the keynote to the effective use of manpower. We must recognize the fact that the ideal use of manpower is impossible, owing to the individual variance of MAN. We order machines, tools, jigs, and fixtures to specifications and we expect these machines

to perform within known limitations. We cannot order manpower to specifications so we must utilize to the highest degree of efficiency the manpower that is available. If we are to succeed in the utilization of manpower, we must be prepared to use all the known means to determine man's probable success in a given job. The industrial physician plays an important role in the utilization of manpower.

Job placement is founded primarily on two basic principles: first, the physical requirements of the job and, second, the physical capacity of the individual. The first principle can be determined by a thorough job analysis or by using the supervisor's knowledge in evaluating the job at the time of each placement. The second principle can be determined only by a thorough medical examination.

The medical examination is the basis of all programs of job placement. By means of this examination an evaluation of the individual's physical ability and disability is used to determine his capacity to perform work. The examination is made keeping four points in mind: (1) medical history, (2) physical examination, (3) laboratory studies including X-ray, (4) personality study, or evaluation of emotional stability.

PHYSICAL EXAMINATION

The objectives of the physical examination are: (1) To acquaint the worker with his physical status; to call attention to physical abnormalities that should be corrected and to stimulate in him the responsibility for the maintenance of his health; (2) to safeguard the health and safety of others by excluding those physically unfit to work or those with communicable disease; (3) To establish a record of the individual's condition at the time of employment with particular reference to existing handicaps, disabilities, or limitations, and (4) to facilitate placement and advancement of workers in accordance with the individual's physical and mental fitness.

In order to be successful, any placement program must have the complete co-opera-

CARDIAC ACTIVITY RESTRICTIONS

Ford Motor Company

NAME _____ SOC. SEC. NO. _____ AGE _____ DATE _____

I. ENVIRONMENTAL

1. NO RESTRICTIONS ☐
2. NO OUTSIDE WORK ☐
3. NO TEMPERATURE EXTREMES (HOT OR COLD) ☐
4. NO HUMID EXPOSURE ☐
5. NO DUST OR FUMES ☐
6. NO WET EXPOSURES ☐
7. NO AFTERNOON OR NIGHT SHIFTS ☐
8. GROUND-LEVEL WORK ONLY ☐
9. LOW NOISE LEVEL ☐
10. LOW EMOTIONAL TENSION ☐
11. FAVORABLE TRANSPORTATION ☐

II. PHYSICAL

1. WALKING - RESTRICTED TO LESS THAN:
 - A. 50% ☐
 - B. 25% ☐
 - C. 10% ☐
 - D. NO WALKING ☐
2. STAIR CLIMBING
 - A. SLOW WITH REST ☐
 - B. NONE ☐
3. SITTING - DOWN
 - A. PART TIME (MINIMUM 50%) ☐
 - B. FULL TIME ☐
4. REACHING
 - A. OCCASIONAL ☐
 - B. NONE ☐
5. STOOPING
 - A. OCCASIONAL ☐
 - B. NONE ☐
6. LIFTING AND CARRYING
 - A. WEIGHT (EXPRESSED IN POUNDS) _____
 - B. FREQUENCY
 1. CONSTANT ☐
 2. INTERMITTENT ☐
 3. OCCASIONAL ☐
 - C. HEIGHT OF LIFT
 1. FLOOR TO WAIST LEVEL ☐
 2. WAIST TO SHOULDER LEVEL ☐
 3. ABOVE SHOULDER LEVEL ☐
7. NO HAZARDOUS MACHINES ☐
8. NO VEHICLE DRIVING ☐
9. NO HIGHLY REPETITIVE WORK ☐
10. ☐
11. ☐

IND. REL.
JAN 37 5123

Fig. 45-1. Cardiac activity restrictions record prepared by physician and medical attendant to record instructions placed on a patient and used for follow-up at regular intervals

tion between the employment department, medical department, and all the supervisors in the plant. The physician, knowing the physical condition of the individual, will endeavor properly to evaluate that physical condition and to approve the employee for all types of work or to restrict him in a way that will limit him to work that is in keeping with his physical handicaps. The supervisor, informed of this limitation, will evaluate the work and will endeavor to place the individual in work that is commensurate with the individual's physical ability.

Classification of Worker with Cardiac Disease

By means of this examination, the individual may be classified as follows:

- Group 1, Unlimited: Acceptable for any type of work in any department,
- Group 2, Limited: If a physical handicap or defect exists which may:
 - 1 Limit an individual's working ability
 - 2 Constitute a hazard at work
 - 3 Be seriously aggravated by certain types of work

An individual with a physical handicap or defect may be classified as *limited* and placed in one or more of the classes outlined in Table 45-1, which are intended to describe

cian. The reason for the restriction is of no practical interest to the supervisor so long as he is informed of the restriction placed on the employee. The restrictions imposed upon the worker because of heart disease may be indicated in a form such as that illustrated in Figure 45-1.

Preplacement and periodic examinations are used for placement purposes as well as for advising the patients in regard to health problems that need attention. These examinations can be used for case finding of various diseases, such as tuberculosis and heart disease.

Some industries have incorporated fairly complete heart studies with routine electrocardiograms whereas others only record electrocardiograms on selected cases or in selected age groups. It has been found to be of great value to run routine electrocardiograms on all patients or at least those over the age of 40 years. This study is of great value for use as a baseline for future reference in case of need for comparison. We have found that our clinical findings are the greatest assistance to us in placing cardiacs on the job.

DISCUSSION

A recent study by a Detroit industrial physician was concerned with 180 individuals who had been diagnosed as having a coronary occlusion with myocardial infarction. Of this group upon recovery, 108 returned to work, 90 returned to their former jobs, 12 returned to lighter work, and only 6 were considered too disabled to work, in spite of the fact that they had been approved to return to work by their personal physician. A follow-up study after a 3- to 6-year period of the 102 cases allowed to return to work showed that 56 were still working, 10 were living but retired due to age, 3 were living but disabled, 6 had quit and were lost so far as follow-up was concerned, 23 had died of heart disease, and 4 died of a disease other than cardiac in nature. It is interesting that almost all died away from work while at rest. In regard to recurrence following the first episode, 60 per cent had no recurrence and

TABLE 45-1 LIMITATIONS AND RESTRICTIONS

Group 2, Limited, seven classes	
Class I	No hazardous machinery
Class II	No heavy lifting
Class III	Ground-level work
Class IV	Avoid dust and fumes
Class V	Avoid skin irritants
Class VI	No extensive walking or standing
Class VII	Restrict to relatively noise-free areas
Group 3, Individuals with a severe handicap that requires special attention for safe placement. These individuals must be placed on a special type of work and remain on that particular job until a special placement is considered in changing them from one job to another.	

in lay terms to the department supervisor the type of work which the employee should avoid. The reason for the limitation to any of these classes is, of course, a confidential matter between the patient and the physi-

40 per cent had one or more recurrences. Seventy-two of the patients were studied regarding their work attendance. Considering an absence of 4 days or more to be significant, it was found that 32 had none for 3 to 6 years, 21 had one significant absence per year, 16 had more than one significant absence per year, and 3 were disabled.

The employability of all persons depends on the emphasis placed on the individual's ability rather than on his disability. It must be recognized that industry employs ability and frequently prospective employees who apply for work have no special ability but do have some problem with their health. In other words, they have no training or skill, and yet they are not qualified physically to do general laboring jobs.

We need more research on some of the basic problems in regard to a practical way to determine what jobs cardiacs can safely perform. There is a golden opportunity to make studies of this type among the large groups of employees in industry today.

Many employers have a great fear or apprehension about hiring employees with heart disease. This fear is based frequently on the liberal interpretation of the compensation laws. Some employers fear great absenteeism and prolonged illness among this group. However, many surveys have shown that employees of this group have excellent records in regard to attendance, quality of work, and labor turnover. This is especially true if the employee is properly placed on the job.

With the progressive aging of the population, heart disease is becoming more common

and, therefore, needs more consideration than ever before. With the dramatic increase in average longevity from 47 years in 1900 to almost 70 years at the present time, we might say that an *Age of Age* is here. If this rate continues, by 1980 approximately 40 per cent of our population will exceed 45 years of age and nearly 26 per cent of the total population of the United States will be in the two decades from 45 to 64. This aging of our work force presents more problems to industry because with advancing age we know there is a higher incidence of diseases of the cardiovascular system.

CONCLUSION

We believe that the individual with cardiovascular disease may be employable. Such employment requires the full co-operation of the patient, the personal physician, the cardiologist, the industrial physician, the employer, and the labor union. The patient must have an examination that evaluates his capacity to work and he must be placed in work with demands that do not exceed his work capacity.

There is need for research to develop better means of evaluating the patient's work capacity.

There is need for revision of legislation which, in some states, practically prohibits the employment of cardiacs.

There is a need for a better understanding of the problem of the employment of the cardiac by the general medical profession, employers, and labor unions.

CHAPTER 46 HEART DISEASE AND FITNESS FOR WORK

*A. Morgan Jones**

The problems encountered in helping patients with heart disease to find and hold suitable employment are determined to some extent by the social, legal, and industrial conditions that exist in the community in which they live. In the United Kingdom the employment of disabled persons is greatly influenced by the "Disabled Persons (Employment) Act, 1944." The aim of this piece of legislation is "to make further and better provision for enabling persons handicapped by disablement to secure employment, or work on their own account." The Act requires the appropriate government department to establish and maintain a register of disabled persons, and lays down the conditions under which a person can have his or her name entered on this register. It requires an employer of a substantial number of per-

* I wish to acknowledge my indebtedness to my associates who have co-operated in this work, and in particular to Dr D. H. Makinson and Dr J. L. Taylor, who undertook most of the clinical work, and to Mr L. A. Briggs, who was responsible for the whole of the social and liaison work. Figure 47-1 is taken from "Services for the Disabled," H.M. Stationery Office, 1955, with permission of the controller of H.M. Stationery Office.

sons to give work to disabled persons on a quota basis, and forbids him to employ an unhandicapped person unless he has his quota of disabled employees. This quota is at present generally 3 per cent but it can be varied by the government from time to time and in special circumstances in individual cases. The Act also makes provision for the financial support of companies formed for the purpose of giving employment to disabled persons, and in 1945 the government set up such a company (under the name Remploi Ltd.), which has factories over the country giving sheltered employment to suitable persons. The Act also made financial provision

recently, under the National Health Service, provision can be made for transport to work in the form of motorized invalid carriages and, in some circumstances, cars, where public transport is not adequate or suitable. In every locality there is a Disablement Resettlement Officer (D.R.O.), whose duty it is to advise disabled persons and to help them to

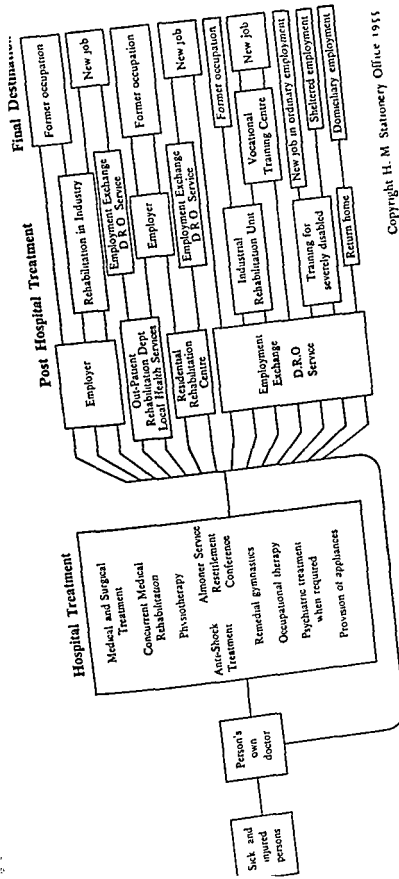


FIG. 46-1 The broad avenues of progress from disability to resettlement in suitable employment

find employment; much of his work is done by creating a close liaison with the hospitals and with the employers and it is his business to bring into operation the facilities provided by the 1944 Act. His success depends to a large extent upon his ability and enthusiasm but fortunately many of these officers have the qualities that make for success in a very difficult and exacting job. In 15 places scattered over the country "Industrial Rehabilitation and Training Centres" have been set up and their purpose is to resettle in employment persons who through illness or other causes have got out of touch with working conditions. They also set out to retrain for new occupations those who have through illness lost the ability to follow their previous employment.

Facilities of this kind (Fig. 46-1) have considerably simplified the task of the physician whose patient has difficulty in following his employment, for there is a well-developed machinery available for finding suitable employment for the patient and for training him in skilled work when this is necessary and possible. So we have been more concerned to find methods of deciding the kind of work that patients with heart disease are fit to do. This involves two problems: first, assessment of the energy cost of various types of work, and secondly, assessment of the work capacity of the patient with heart disease. During the years 1948-52 my department, in association with the Departments of Occupational Health and Physiology, attempted such a survey.

THE ENERGY COST OF CERTAIN INDUSTRIAL OPERATIONS

Around Manchester we have a wide variety of occupations, for the traditional dependence of Lancashire upon the cotton industry is a thing of the past. Thus Turner,¹ who undertook this work in association with my Department, was able to assess the energy cost of varied operations in chemical industries, storage battery works, light and heavy engineering, plastics, office machinery operators, watch and clock repairing, tailoring,

machinists, boot and shoe repairing, etc. His detailed observations have been published. He followed the scale of metabolic work used by Orr and Leitch² but converted it to total metabolism per square metre of body surface, instead of excess metabolism over resting level for an "average" man of surface area 1.77 square metres. On this basis the various operations were divided into four groups.

Light work. Up to 90 calories per square metre per hour.

Moderate work. 90-134 calories per square metre per hour.

Heavy work. 135-220 calories per square metre per hour.

Very heavy work. Over 220 calories per square metre per hour.

Turner's objects were, in particular, to provide clinical workers with information about the relative severity of a variety of occupations in the area and to try to decide whether in assessing the severity of work, it was necessary to measure the energy cost or whether assessment by simple observation of the work was accurate enough for practical purposes. A separate assessment of each process was therefore, made by simple observation of the work and the process classified into light, moderate, and heavy or very heavy work.

Work classified by simple appraisal: heavy or very heavy turned out on measurement to involve an energy expenditure of between 120 and 230 cal/sq m/hr. Work that seemed to be moderate varied between 70 and 130 cal/sq m/hr and apparently light work ranged from 55 to 80 cal/sq m/hr. An observer with some experience of watching industrial operations can therefore classify the work with a fair degree of accuracy. Two qualifications must be made, however. It is essential to appraise the actual process, for working methods may so differ in the same process in two factories that the energy cost may be considerably different; secondly, in work requiring intermittent effort, the estimation of average oxygen uptake may give a falsely low assessment of peak efforts, and in this instance simple appraisal may be better than actual measurement of average energy cost.

ASSESSMENT OF THE CAPACITY FOR WORK

I believe this to be a very difficult problem. It has been the practice to assess capacity for work by the ease with which effort evokes symptoms. This is the basis of the classification introduced by the New York Heart Association, which has achieved international acceptance.

- Class I No limitation; ordinary activity causes no discomfort.
 Class II Slight to moderate limitation, ordinary activities cause discomfort.
 Class III Considerable limitation, unable to undertake ordinary activities
 Class IV Symptoms at rest.

If we classify the energy cost of various jobs as

- Group A Heavy or very heavy work, more than 135 cal/sq m./hr
 Group B Moderate work, 90-134 cal/sq m./hr.
 Group C Light; up to 90 cal/sq m./hr

then it might be expected that Class I patients might do Group A work; Class II patients might do Group B work, Class III patients might do Group C work, Class IV patients are not generally employable

TABLE 46-1. COMPARISON OF SYMPTOMS WITH "EXPECTED" SUITABILITY OF WORK

Functional class	Symptoms at work			No symptoms at work		
	Group A	B	C	Group A	B	C
I	6	1	0	9	53	9
II	2	2	0	1	24	1
III	2	5	0	1	0	3

In fact, this expectation does not work out too badly in practice. In my clinic 125 employed patients were studied on this basis. One hundred one were free from symptoms at work and 24 had symptoms at work. In Table 46-1 their actual work is compared with the predicted work on the basis sug-

gested above. The groups joined by diagonal lines are those in which the actual work would be regarded as suitable on the basis suggested. Figures above the diagonal lines indicate that the work is lighter than predicted; below the diagonal lines the work is heavier than predicted. Of 63 patients in work lighter than predicted only one had symptoms, whereas when work was heavier than predicted 16 of 18 patients had symptoms (Table 46-2).

TABLE 46-2. RELATION OF WORK TO SYMPTOMS

Work	Number of cases	Number with symptoms at work	Per cent with symptoms at work
Lighter than predicted	63	1	1
"Correct"	44	8	18
Heavier than predicted	18	16	89

In cases "correctly" placed the position is far less satisfactory for 8 of 44 had symptoms at work. It may be that the standard is too high, and it is notable that 64 of 125 patients had obtained work lighter than this system of prediction would have indicated.

Other Cardiac Factors in Assessment

Although since the days of Mackenzie it has been generally accepted that the severity of heart disease is best judged by the patient's capacity for effort, Mackenzie himself laid down certain qualifications, for he realised that in active cardiac lesions, such as acute rheumatism or a recent coronary thrombosis, exercise may do harm although the patient may be able to undertake it without any distress. Unfortunately, in recent years it has become plain that it is often very difficult to be sure when ischaemic or rheumatic heart disease is in an active phase. In rheumatic heart disease a comparatively high proportion of auricular biopsies taken at valvotomy show evidence of active carditis and there is evidence that the presence of such changes in the auricle is a good indication of similar changes in the ventricles. Similarly, in ischaemic heart disease the rec-

ognition that acute events in the coronary arteries are not only manifested by the clinical syndrome of *coronary thrombosis* but also by far less dramatic symptoms has led to the division of ischaemic heart disease into acute and chronic phases (which might be better named *active* and *quiescent* phases) instead of the older subdivision of *coronary thrombosis* and *angina pectoris*.⁸ It has also emerged that in obstructive valvular lesions very severe heart disease may be present with little in the way of symptoms. For instance, a boy with pulmonary stenosis, seen recently in my Department, had a systolic pressure in the right ventricle of 280 mm Hg but he had an almost normal pulmonary artery pressure, a normal heart output, and was entirely free from symptoms, even when playing football. In aortic and mitral stenosis, very similar patterns may be seen.

There is, therefore, no short cut to the assessment of the patient with heart disease: no simple inquiry about the capacity for exertion will serve to avoid a full and exhaustive investigation of the patient. In the end, fitness for work can only be judged as an individual matter in each case, and each piece of information gained from the history, examination, and investigation is only one brick to be used in building the final picture. This is one reason why we have failed to find any very satisfactory correlation between factors like cardiac size and vital capacity with

TABLE 46-3 PERCENTAGE OF PATIENTS WITH RHEUMATIC AND ISCHAEMIC HEART DISEASE IN EMPLOYMENT

	Employed	Unemployed
Rheumatic	96 (79%)	25
Ischaemic	9 (20%)	36

the ability to work. It has been pointed out that there is some correlation between the etiology of heart disease and the ability to work; Kresky and Goldwater⁶ found that 70 per cent of male patients with rheumatic heart disease were working, as contrasted with only 26 per cent of patients with degen-

erative heart disease. Our own figures are similar (Table 46-3).

This difference may be partly a matter of age, for taking all our cases together many more of the younger patients were in employment, irrespective of the type of heart disease (Table 46-4).

TABLE 46-4 PERCENTAGE EMPLOYED IN VARIOUS AGE GROUPS

Age	Employed	Unemployed
Less than 35	75 (83%)	15
35-55	41 (54%)	35
Over 55	9 (32%)	19

Increasing disability with age is an important though not the only factor in reducing the proportion employed in the older age groups (Table 46-5).

TABLE 46-5 RELATION OF DISABILITY TO AGE

Age	Functional class		
	I	II	III-IV
Less than 35	65	29	9
35-55	19	56	44
Over 55	3	12	13

EXTRACARDIAC FACTORS INFLUENCING EMPLOYMENT

The relation of age to employment leads to some consideration of the extracardiac factors that influence employability, for these are probably at least as important as the cardiac condition. We have studied these in a clinic organized for this purpose and 322 patients were under supervision for periods up to three years. These patients were referred to this clinic in three ways:

1. From the Ministry of Labour, as registered disabled persons said to be suffering from heart disease (111)
2. Male patients referred from other clinics in the Hospital (151)
3. Patients, seen in my Department concurrently with the survey, in need of help with employment (60)

The source of these patients is important, for most of them were seen because there was considered to be some employment problem, either by the Ministry of Labour, other clinics, or myself or my associates in our Department. It is to be expected, therefore, that employment difficulties will be the rule, and will exceed in number those in a similar group of unselected patients with heart disease.

It was necessary to exclude 72 of these patients from the analysis either because they were found to have no heart disease (36), owing to failure to attend regularly (35), and one patient who was not of employable age. The group with the highest percentage of rejections because no heart disease was found was that referred by the Ministry of Labour as Registered Disabled Cardiacs (26 of 111).

The remaining 250 patients were fully surveyed clinically, and followed up regularly both by a clinician and by a social worker who recorded particulars of any changes of work, and other social details. He also interviewed employers, and developed a close liaison with them, with their medical services and with the Ministry of Labour services. Of

120 Men Unemployed at Beginning of Survey

Of the 120 unemployed, 92 (77 per cent) found work. Of the remaining 28, only 9 were still trying to obtain work at the end of the survey, 13 were too ill to work, and 6 were dead. But of the 92 who obtained work, 31 did not hold it and of these only 9 continued to seek work, 11 became too ill to work, 9 died, and 2 retired. Of the 61 who appeared to be satisfactorily resettled in work at the end of the survey, 42 had been at work for over 12 months. Of the 92 who found work, 47 did so through the efforts of the DRO (Disablement Resettlement Officer), 9 were placed directly by the clinic, and 36 found work by their own efforts.

Factors Influencing Resettlement in Work

The most important factors were age, duration of unemployment, stability of previous work history, personal attributes, and financial considerations. Although we have, as part of our education in this subject, studied these factors fairly fully, they deserve only brief consideration here, for this problem is not peculiar to heart disease but exists in all forms of disablement, and has been widely studied.

AGE Excluding patients who died, it was found that satisfactory resettlement was achieved in 41 of 56 patients under 45 years of age, but in only 20 of 49 over this age. Of 22 patients who had no work at all during the survey, 18 were over 45 years of age.

DURATION OF UNEMPLOYMENT Of 61 patients satisfactorily resettled, 32 had been unemployed for less than 6 months, and only 10 for more than 2 years. Of the 22 who were unable to find any work, only 3 had been unemployed for less than 6 months and 10 had not worked for over 2 years (Table 46-7).

STABILITY OF PREVIOUS WORK HISTORY. It is difficult to express this in simple figures, but the most useful indication seems to be the number of jobs a man has held; unfortunately in the present survey this had little meaning for during much of the relevant period wartime regulations prohibiting free change of work were still in force. In fact 25

TABLE 46-6 EMPLOYMENT CHANGES IN 250 PATIENTS IN 3 YEARS

Remained in same employment	71
Work changed or adjusted	44
Lost or gave up work and not re-employed	15*
Unemployed throughout	28*
Work found and remained in employment	61
Work found but later lost	31*
Total	250

* These groups include 15 who died

the 250 patients, 25 had congenital heart lesions, 160 acquired rheumatic or (rarely) syphilitic heart disease, and 65 had ischaemic heart disease. At the beginning of this study, 130 were employed and 120 unemployed. When the final survey was made 3 years later 176 were employed, 59 unemployed, and 15 dead. The employment changes that occurred during the three years of the survey are summarized in Table 46-6.

(41 per cent) of the 61 resettled men had had more than 6 jobs during their life, but of 22 who found no work 12 (55 per cent) had had more than 6 jobs. The circumstances of the survey thus prevent any satisfactory conclusion on this point.

TABLE 46-7 PERIOD OF UNEMPLOYMENT AND RESETTLEMENT AT WORK

Period unemployed	Number	Number resettled	Temporary work	No work
Less than 6 months	43	32	8	3
6 months to 2 years	40	19	12	9
More than 2 years	22	10	2	10

PERSONAL ATTRIBUTES Mental ability and social standards both have some influence on the possibilities of resettlement but our figures are not large enough to provide convincing evidence on these points. The attitude of the man to his work and to his heart disease is, however, a sufficiently important factor to be clearly demonstrated in the number of cases available. This attitude was placed in one of 4 categories

- Good* Actively seeking work and co-operative in attempts to help him
Fair Still with the will to work, but doubtful of his ability to do so
Poor Not seeking work, and maintaining that he had difficulty in working
Bad Obstructive of attempts to find him work, and claiming that his heart made him unfit to work.

Resettlement in these categories is summarized in Table 46-8

TABLE 46-8 ATTITUDE TO WORK AND HEART DISEASE

Attitude	Number	Resettled	Temporary work	No work
Good	52	34	12	6
Fair	29	20	2	7
Poor	21	7	6	8
Bad	13	0	2	1

Thus of those with a good or fair attitude to work two thirds were resettled, if the attitude was poor only one third, and none was satisfactorily resettled when the attitude to work was bad.

FINANCIAL CONSIDERATIONS These were studied in some detail and include civil status, number of dependents, and other members of the family contributing to the family income. Although the results suggest that financial need to work plays a part in successful resettlement, the differences are not large enough to be very significant. The need to support a family is obviously a considerable stimulus to obtain work, but this operates much more in skilled workers who can substantially increase the family income. On the other hand the unskilled worker with a large family, and consequently large family unemployment allowances, may gain very little financially from work. This only applies, however, to those with the lowest earning capacity and, for the majority, unemployment is a very serious hardship.

The reasons for difficulty in finding employment may be summarised by considering the 44 men in this group who were unemployed at the end of the survey. The prospects of employment in these men were regarded as good in 4, fair in 4, poor in 30, and there was no prospect of work in 8. The primary reasons for the poor or negligible prospects of work in 38 are summarized in Table 46-9.

TABLE 46-9 REASONS FOR POOR OR NEGLIGIBLE PROSPECT OF RE-EMPLOYMENT

Severity of heart disease	17
Mental attitude to work	10
Age	6
Lack of opportunity or experience	3
Low mental ability	2
Total	38

Thus incapacity to work owing to the severity of the heart disease was the primary reason for unemployment in less than half the cases, and mental attitude to work and age were both important factors.

130 Men Employed at Beginning of Survey

These men presented comparatively minor problems, and 71 continued in the same work without any major adjustment throughout the period of study. Forty-four changed their work, or major adjustments were made in it, often on medical advice. Of the remaining 15, 11 were dead when the final assessment was made, 2 were ill, one was seeking work, and one had retired. For the purpose of comparing this group with the unemployed group and for reviewing the reasons for major adjustment or change of work it is, therefore, proposed to consider only the 115 men working at the end of the survey.

Comparison with Unemployed Group

AGE This has already been considered (Table 46-4). Of 115 employed 63 per cent were under 35, and only 16 per cent over 45 (compared with 29 per cent under 35 and 45 per cent over 45 in the unemployed group)

STABILITY OF PREVIOUS WORK HISTORY The difference between the two groups was striking (Table 46-10). Age may have been

TABLE 46-10 STABILITY OF WORK HISTORY IN EMPLOYED AND UNEMPLOYED GROUPS

	Number	1 job	2-4 jobs	More than 4 jobs
Employed	115	24	83	8
Unemployed	105	6	25	74

a factor in the smaller number of jobs in the younger employed men but skill is probably more important (Table 46-11)

TABLE 46-11. COMPARISON OF SKILL IN EMPLOYED AND UNEMPLOYED GROUPS

	Number	Skilled	Semiskilled	Unskilled
Employed	115	55	20	40
Unemployed	105	23	19	63

PERSONAL ATTRIBUTES The attitude of those employed to work was naturally better than in those unemployed (Table 46-12)

Mental ability and social standards were

TABLE 46-12 COMPARISON OF ATTITUDE TO WORK IN EMPLOYED AND UNEMPLOYED GROUPS

	Number	Good	Fair	Poor	Bad
Employed	115	106	7	2	0
Unemployed	105	52	29	31	3

generally better in the employed group (Table 46-13)

TABLE 46-13 COMPARISON OF MENTAL ABILITY AND SOCIAL STANDARDS IN EMPLOYED AND UNEMPLOYED

Characteristic	Employment status	Number	High	Average	Low
Mental ability	Employed	115	38	61	16
	Unemployed	105	6	64	35
Social standards	Employed	115	23	91	1
	Unemployed	105	6	69	30

CHANGES AND MODIFICATIONS OF WORK

In 53 of 130 cases there was some important unsatisfactory feature of the work, this ranged from excessive travelling, to long hours or too strenuous work. In 30 of these cases these factors were regarded as sufficiently important to justify advising a change of work. In the end, this advice was followed in 19 cases (3 of these men died during the survey). In addition, 27 of the 77 men in satisfactory work changed it for a variety of reasons that included training, promotion, chance, and preference for another job for reasons unconnected with the heart disease. It is often very difficult to discover why a man changes his job, and one sometimes wonders if he knows himself!

A man was never told lightly he must change his job, this was advised only after careful consideration and only when it seemed essential following a full discussion by all the members of the clinic staff and full assessment of the pros and cons with the patient. Then either the man himself or the clinic social worker approached the employer. A very satisfactory degree of co-operation was exhibited by almost all employers and the

majority of the men were resettled with the same employer. A few obtained work with another employer, naturally, this was more likely to occur in the case of younger men who were more acceptable to a new employer and had less claim to consideration as old employees. Two examples may illustrate the sort of problem that has arisen in our experience.

A man, aged 47, had been with a firm of cable makers for 24 years and was a charge hand when he first came to the Clinic. His work involved considerable physical labour including the manual mixing of materials, which required some skill and experience but clearly overtaxed him. He was dubious of approaching the management but agreed to do so if the Clinic social worker undertook to confirm to the employer the necessity for adjustment of his work. His employer willingly found him routine clerical work, which he continued to do very well until the end of the survey.

A boy of 17 had been apprenticed for 2 years as an engineer. His work involved the dismantling of cement mixers but he would later become a maintenance engineer, travelling the country and often being required to manhandle the mixers. With the consent of his parents an approach was made to the firm, who confirmed that the boy's future work would involve heavy exertion and suggested that he was in any case unsuited to the work. In view of this the boy was resettled, with the help of the Youth Employment Bureau, as an improver maintenance electrician, a job that he preferred and was well within his capabilities.

In our experience it is wise to consider carefully before advising a man to change his work, for it may well unsettle him and will, at least temporarily, raise doubts in his mind concerning his ability to earn his living. For this reason 23 men with work that was unsatisfactory to some extent were not advised to change their occupations. Many of these were engaged in highly skilled or very specialised work, and in some of them it was clear that the work would become more responsible and less arduous in the future. A

man of 28 was employed as a shot-firer in a coal mine. He was obviously a man of intelligence and ability who was well on the way to becoming a Deputy. It was likely that within a few years he would gain an administrative or technical post and it seemed better not to unsettle him, although he had some dyspnoea at work. Three years later he had gained his Deputy's certificate and was in line for further promotion.

CONCLUSIONS

In the United Kingdom reasonable statutory provisions exist to facilitate employment of the disabled. These provisions include the legal obligation of all large employers to engage disabled men on a quota basis, liaison services (by the DRO's) between the medical services on the one hand and, on the other hand, the State rehabilitation or re-training facilities and the employers. The medical services are, therefore, primarily concerned with assessing the ability of the patient to work, and in encouraging him to do so. But it is important for the physician to be conversant with the sort of industrial operations that his patient might undertake. In assessing the energy cost of industrial operations, simple appraisal by a practised observer can rival or, in some cases, excel physiological assessment by the measurement of actual energy expenditure. A survey by both methods of a wide range of industrial operations in the area served by the clinic has proved useful background information in resettlement.

In assessing ability to work, no single factor can be regarded as of paramount importance, though the most useful simple method is the functional classification of the New York Heart Association. Objective measurements, such as the vital capacity, have a more limited usefulness. The liability to an active phase in rheumatic and ischaemic heart disease is an additional factor that must, in individual cases, be taken into account. In practice, there is no satisfactory alternative to a full assessment of all the factors that play a part in the cardiac condition and this is an

indispensable preliminary to intelligent resettlement in work.

In addition to the cardiac factors that influence employability, there are equally important extracardiac considerations. Of these the more important are age, duration of unemployment, stability of previous work history, personal attributes, especially attitude to work and financial considerations. Age and attitude to work may play a dominating part in employability.

If a man is already in employment, change of work should be suggested only after very careful consideration, especially if his present employment is of long standing. Suggestions of this kind can undermine a man's confidence in his ability to earn his living, and may seriously unsettle older men, who usually continue in their original employment or give up altogether. In our experience, employers have been co-operative and their legal obligation to employ a quota of disabled men exerts a useful pressure upon them to provide alternative work for old employees.

One of the more important fields of work, not considered here but of considerable practical consequence, is the special employment problem of disabled young people. This has been studied by Ferguson, Macphail, and McVean,⁷ who pointed out how loss of education through illness in childhood may force youths into unsuitable manual work, and stressed the danger that unguided choice of work in adolescence may lead to subsequent unemployment with its demoralizing effect at this age. Physicians may add their quota to the burden of the handicapped child by unwarranted restriction of activity, which may lead to a lifelong fear of ill effects from all forms of physical activity. The transition from childhood to adult life is thus a period with its special hazards for those with heart disease, and it is at this time that most can be done by guidance in the choice of suitable work and by providing appropriate training facilities.

Finally, I think the most useful thing we have gained from studying these problems is

an attitude of mind. Facilities for assessment of patients with heart disease and an organization for training and placing them in suitable employment are indispensable tools for this work, but they cannot replace a personal and lasting interest in the welfare of each patient by his physician on the one hand, and his employer on the other. Physical ability to work is no guarantee of success in resettlement, nor is serious physical handicap an inevitable bar, for the man's will to work may be a dominating factor. Some patients with heart disease need little help to enable them to become or to remain self-supporting and useful members of their community, others, despite every effort to help them, and possibly considerable physical ability to work, remain useless and dependent upon the support of the community. But between these extremes there are many who can be helped to regain their livelihood and self-respect by the wise use of proper facilities for the work of resettling them, and by the real interest and serious endeavour of physicians, social workers, and employers who believe this work to be worthwhile.

BIBLIOGRAPHY

- 1 TURNER, D. The energy cost of some industrial operations, *Brit J Indust Med* 12 237, 1955.
- 2 ORR, J. B., and LITCH, I. Determination of caloric requirements of man, *Nutrition Abstr & Rev* 7 509, 1938.
- 3 MACKENZIE, JAMES. *Diseases of the Heart* 3d ed London, H. Frowde, 1913, p. 363.
- 4 MCKEOWN, F. Left auricular appendage in mitral stenosis, *Brit Heart J* 15 433, 1953.
- 5 PROGER, S. Acute and chronic phases of coronary atherosclerosis, *Mod Concepts Cardiovas Dis* 20 96, 1951.
- 6 KRESKY, B., and GOLDWATER, L. J. Occupational potentialities of cardiac patients, *Am Heart J* 27 623, 1944.
- 7 FERGUSON, T., MACPHAIL, A. N., and McVEAN, MARGARET L. *Employment Problems of Disabled Youth in Glasgow* Medical Research Council Memorandum No. 28 London, Her Majesty's Stationery Office, 1952.

REPORT OF PANEL ON WORK CLASSIFICATION

Leonard Goldwater

I would like to preface my remarks as some of the other moderators have done this morning by expressing the appreciation and admiration of the members of our panel for the arrangements made here by Dr. Belknap and his colleagues. As a result of the papers presented, and the discussions which followed, the panel on work classification wishes to present the following report.

The field of interest of those concerned with work classification includes two major areas. First, a study of the effects of employment on the course of heart disease and its obvious converse, the effects of heart disease on employment, and second, the methods of improving the employment opportunities for cardiacs. This includes both the acceptance and the retention of cardiacs in gainful employment. These concepts have been stated before and were unanimously reconfirmed by the panel as still being valid.

We will direct our attention, first, to the effects of employment on heart disease. While a small amount of information is available on this subject in the medical literature and some additional data were presented at this

conference, the panel felt that a need for further facts still exists. Research studies that can yield the desired material must be carried out over long periods of time, perhaps as long as ten to twenty years. Unfortunately, sustained financial support for such studies is difficult to obtain. A number of groups, some of which were represented on the panel, have already collected a limited amount of information. This Conference represents the first time that this material has been brought together from all parts of the country. Additional data were brought to us by Dr. A. Morgan Jones, our guest from England. It can be stated now that hundreds of cardiacs representing thousands of person years of productive activity have been observed in a wide variety of jobs with no evidence of adverse effects on the course of their heart disease. In fact, it can be stated that there is substantial evidence that in many cases employment actually resulted in improvement, not only in emotional status but also in physical condition. The individuals observed included some with heart disease existing prior to employment and some for whom employ-

ment had been retained and who returned to work following a heart attack. It should be pointed out that persons upon whom these observations were made were placed or kept at work through various Work Classification Units. To achieve these results two simple conditions had to be met. First, employment was selected with reasonable intelligence, and second, the subjects remained under adequate medical supervision. It would be extravagant, of course, to maintain that all cardiacs may continue in employment or that all types of work are suitable for those having heart disease. On the other hand, evidence has been presented that, under the conditions mentioned above, a vast majority of cardiacs can work and that there are relatively few types of occupation that need be interdicted. No evidence was found that work per se was a primary cause of heart disease. Among the cardiacs who have been unsuccessful in securing or maintaining employment, there are many whose failure is due to numerous factors other than heart disease. Emotional disturbances, mental attitude, age, lack of skill or experience, lack of jobs, low mental ability, as well as many other illnesses, are some of the noncardiac reasons for employment difficulty. Factors such as these influence the employment status of all segments of the population as well as those with heart disease.

The question of return to work following a severe attack of ischemic heart disease was given special attention in our discussions. Incontrovertible evidence has been adduced to show that of those who survive the attack many, if not most, are able to return to their original jobs with safety. A change of job

or work. Evidence was presented to the panel that many individuals sustain myocardial infarcts with manifestations that are so mild that no loss of time from work occurs. In some of these cases that were discovered in the course of routine electrocardiographic studies, it was even impossible to elicit any of the symptoms of ischemic heart disease. This,

incidently, is in line with what we just heard in the report of Dr. Edwards from the panel on pathology about the discovery of infarcts of the myocardium in persons dying of other causes and in whom no history of heart disease was obtained.

The panel spent a considerable amount of time discussing *Work Classification Units*. Reports were presented by directors of several of the more successful units and a summary was presented covering the experience of 15 additional units. These reports permitted the panel to arrive at a number of conclusions. There was complete agreement among the panelists that the *Work Classification Unit* as originally conceived at Bellevue Hospital in 1941, and subsequently developed and improved by others, is an important and practical device for improving the employment opportunities of cardiacs as well as for studying the effects of employment on the course of heart disease. The evidence presented in the first part of this report is based largely on data collected at a number of work classification units. The *Work Classification Unit* approach has been adopted by a few medical departments in industry and perhaps elsewhere. It is safe to say, however, that the full potential of this type of program has not been developed. The panel agreed that there is a place for additional work classification units as well as for additional applications of the idea in industry, in union health centers, other cardiac clinics, in rehabilitation facilities of all types, and by individual physicians. Such expansion, if properly carried out, can be expected to yield much needed information on the effects of employment on the course of heart disease and, at the same time, to improve employment opportunities for cardiacs. The stipulation *properly carried out* implies, among other things, that provisions be made for long-range follow-up and for the correlation of the course of the disease with occupational factors. The panel felt strongly that no new work classification units should be established without proper preliminary study as to community needs, without adequate preparation, and without assurance of an adequate staff and financial support. It

was made clear to the panel that a number of work classification units presently existing may be characterized as being marginal or substandard. This suggests the desirability of establishing minimum standards and of developing methods for up-grading those units which fail to meet these standards. There was some feeling that there is no justification for the existence of a substandard unit, particularly since such a unit may tend to discredit the whole *Work Classification Unit* idea.

The possibility of setting up a central agency to which all work classification units might report their results was discussed by the panel. Assuming the adoption of uniform records and reports, such an agency would make possible the pooling of data with the obvious advantages to be derived therefrom. It was felt that the American Heart Association would be a logical organization to develop such a program.

The educational possibilities of the *Work Classification Unit* idea were discussed in our meetings. Evidence was presented to the panel that work classification units and activities arising from them have had an appreciable effect on the thinking of many individuals and groups. This has been accomplished through direct participation in the activities of the unit, by the publication of reports on research studies, lectures, articles, lay publications, cardiac-in-industry conferences, seminars, and other similar meetings. It was agreed, however, that much more must be done to demonstrate to physicians, employers, employees, and the public generally that cardiacs can work. The suggestion was made that a work classification unit should be connected with every medical school, since most of the patients of future doctors will be employed persons, and since diseases of the heart will be among the most frequent ailments the doctor is called upon to handle. Among the most important educational possibilities of a work classification unit is its value as a demonstration program. The demonstration includes both theory and practice. The panel felt that the functions of demonstration can be carried out best if the

unit has considerable independence and freedom to develop those relationships which are necessary for effective action. This does not mean that it should exist in a state of isolation, but rather that it should be free to establish its own policies on such questions as acceptance of referrals, financing, hours of operation, staffing, and other similar matters.

In its discussion of possible future developments the panel felt strongly that since problems relating to the employment of cardiacs are national in scope and of major proportions, agencies that operate on a nation-wide basis must assume increased responsibility for future developments. There is a proper place for both voluntary and governmental groups to be concerned. Greater financial support is needed to expand the research, educational, and service activities in the field of work classification and selective placement of cardiacs. The matter of proper vocational guidance and vocational training of the adolescent cardiac has scarcely been touched. Widespread dissemination of information such as that brought out at this conference would be most desirable, using various medical and nonmedical media. Intensified efforts to educate physicians on matters of heart disease and employment are surely needed. Other groups such as employers, social workers, vocational counselors, personnel directors, officials of labor unions, nurses, and rehabilitation workers must also be included in the educational efforts.

The First Wisconsin Conference on Work and the Heart has made it possible to bring together the best and the latest information in this field and the most competent observers working in this area. This Conference will

DR. RODNEY BEARD: I want to comment on the preceding report, not as a moderator of a panel but as an individual. Dr. Goldwater hinted at the incompleteness of cardiac work classification units in terms of rehabilitation because of their lack of adequate vocational counseling and social work. I submit that cardiac work classification units are often incomplete, lacking the staff for pro-

longed rehabilitation counseling and social work, since these units often are basically diagnostic centers rather than rehabilitation centers. I wonder if there are any compelling reasons for keeping rehabilitation of cardiacs separate from rehabilitation of people with other kinds of disorders. Are we to go on and have specialized rehabilitation units and work classification units for diabetes, for arthritis, and for all the other classifications that might be so concerned? I think I will leave that as a question that may or may not be answered here this morning.

DR. A. MORGAN JONES: I would like to say, first, how much I have enjoyed this conference and how grateful I am to Dr Goldwater and the panel for giving me such an interesting time. I have learned a great deal here.

I am a very strong believer in the idea of the work classification unit, and I feel there is room for a great deal more effort in this social way, in the social aspects of heart disease, certainly in England, and I imagine here, too. The thing I am not quite so happy about is whether it should also be segregated from other aspects of other problems in heart disease. I am sure work classification units are essential for development and for research and as an example of how the job should be done. However, I would like to see the ideas developed in them permeate more extensively into the routine clinical work in cardiology everywhere and in all cardiac clinics. Their success really depends to some

extent on this, because I doubt very much whether they can serve as work classification units to the whole population of any country. This is my interpretation of their purpose with their experience to all those who work in this field. This would be my ideal of the way the work classification units should go in the future with the preservation of a considerable number of high-grade centers that can set standards and develop the research aspects of the problem as well.

DR. LEO PRICE: Dr Jones has given me courage to make a few additional remarks concerning classification units. I believe their value and effect in industry have not been emphasized as much as they should be. The material you can accumulate would be far more valuable within industry than within classification units. I think the value of the classification units that will, perhaps, be added to industrial groups will be very helpful. I think it is only in those large corporations that have a large number of employees that we are going to find out, as we did in our industry, that 35 per cent of the ambulant patients had coronary occlusions and that 25 per cent of them lost no time whatever from their work. One more thing I wanted to add is that, while you mentioned three tests for fitness, fitness to work, a test for the impairment of the heart, and a test of disability, I believe we omitted a test of productivity. The employer is going to settle this question.

PART **V** WORKMEN'S COMPENSATION

CHAPTER 47 MAGNITUDE OF THE PROBLEM OF THE CARDIAC-IN-INDUSTRY

Harry E. Ungerleider and Richard S. Gubner

INTRODUCTION

The problem of heart disease in industry may be stated very concisely. Persons with heart disease can work, do work, and should work. Their employment should be under medical guidance without prejudice or restriction other than that their jobs not be hazardous to themselves or others, nor impose unreasonable liabilities upon employers and auxiliary agencies, private or governmental, which assume a share of responsibility for their welfare. Like all principles, this is clear and simple. But between principle and practice there is a vast breach, and it is our purpose to explore this breach. That a problem and a serious one exists is not basically the fault of the patient with heart disease, nor the physician. Rather, it is a social and financial issue, one which has arisen from inadequate or misguided legislation, and an understandable indisposition on the part of industry to assume unwarranted potential financial risk. I may stress now, and will

illustrate later, that the unhealthy state of affairs that now exists can be remedied, quite literally, by calling in the doctor. I have been asked to survey the magnitude of the problem. This may be approached in two ways, one to indicate the extent of heart disease in the industrial population, and two, to indicate the extent of interest and attention the problem is currently receiving.

INCIDENCE OF HEART DISEASE IN INDUSTRY

It has become almost trite to preface discussions of heart disease with imposing figures of its frequency, but this is the very key to the magnitude of the problem of heart disease in the industrial population. It is estimated that some ten million of the United States population have some recognizable form of heart disease. Beyond this, there is a vast reservoir of heart disease, unrecognized or unrecognizable with present methods. In the study conducted by White, Edwards, and Dry¹ at the Mayo Clinic a few years ago,

comprised of an autopsy investigation among persons dying of causes other than heart disease, it was found that a severe grade of coronary arteriosclerosis was present in well over two thirds of persons above the age of 50. Similarly, in a clinical study which we conducted among a series of 320 insurance applicants in the Diagnostic Laboratory of the Equitable Life Assurance Society,² we found evidence of arteriosclerosis in fully half of the subjects between the ages of 51 and 55 years, and in 75 per cent of the cases above the age of 55 years. By far the most common sign of arteriosclerosis was calcification of the abdominal aorta as seen in x-ray studies. Additional abnormalities were revealed by x-ray studies of the chest, including roentgenkymography and electrocardiography, the electrocardiogram being abnormal in 9 per cent of the group studied. The age group included in this investigation is precisely that of the industrial population and, in fair measure, may be considered to reflect the prevalence of arteriosclerosis in the industrial population. Now, of course, the enormous prevalence of arteriosclerosis does not signify that this disorder assumes clinical significance in all or even more than a fraction of the population. However, taking a single complication of arteriosclerosis, coronary occlusion, which is a type of heart disease occurring predominantly among males during their working years, it has been indicated by Master and his associates,³ using samplings of New York State death certificates for 1942, that there occurs in the nation as a whole about eight hundred thousand attacks of acute coronary occlusion yearly. Adjusting for population trends in the twelve years intervening since this study, it must reasonably be concluded that the number of attacks of coronary occlusion yearly now pass the one million mark. As pointed out by Kline,⁴ if even half of these survivors return to work, which is most certainly the case, some five hundred thousand persons with coronary disease of clinical significance are being added yearly to the labor market. This, then, is the situation for one type of heart disease alone. If other forms of heart disease

are considered, such as hypertensive heart disease, a similar array of imposing numbers may be cited. To this may be added the mitral heart disease, congenital heart disease and less common forms of heart disease such as syphilitic heart disease, cor pulmonale, myocarditis, etc., all occurring in small numbers, it is true, but still of real significance. Several of these, although not important in the total, assume particular significance in individual industrial situations. An example is cor pulmonale, as it occurs in workers in fields carrying occupational hazards to the lungs.

EXPERIENCE WITH CARDIAC WORKERS

Let us take a specific illustration of the problem by considering the experience of a single large industrial organization that includes a wide diversification of jobs, the Consolidated Edison Company of New York. Franco⁵ has recently reported the results of a cardiovascular survey covering the nearly 25,000 employees of Consolidated Edison for the year 1952. In periodic examination of apparently normal persons over 40 years of age who had no medical complaints and no apparent symptoms, evidence of heart disease or conditions potentially capable of producing it, such as hypertension, were found in 15 per cent of those examined. Significant cardiovascular disease was found in 7 per cent, almost half this number, a figure corresponding closely with an earlier survey made at the Eastman Kodak Company.⁶ In 36 per cent the heart disease was of sufficient degree to require medical attention. In the total of 896 cases of significant heart disease, coronary artery disease led in frequency and accounted for 58 per cent of the group. Hypertensive vascular disease was next in frequency, occurring in 32.5 per cent of the cases. Rheumatic heart disease was present in 7.2 per cent of the total group. Franco points out that the relatively low incidence of rheumatic heart disease is probably attributable in part to careful pre-employment screening in former years, thereby excluding many persons with rheumatic

valvular lesions. This aspect of exclusion of cardiacs from employment is at the very core of the problem of heart disease in industry, and one to which we will return presently. Some further findings in the Consolidated Edison study are of interest. Fifty-four per cent, more than half of the total, had an unblemished attendance record over a period of a full year. The rate of absenteeism of those employees with heart disease was approximately 25 per cent below the general average. On the other hand, the disability and severity rates were above the company average. Approximately 30 per cent of those with significant heart disease were able to hold their regular full-time jobs without any physical restriction. Of the remaining 70 per cent who were on limited duty, about half retained their regular jobs, while the others were transferred to lighter duty. A point of particular interest in this organization with a stable working population was that the majority of workers with cardiovascular disease had been on the job for over 25 years. The high point came at about the age of 55 years, when the average length of service had reached 31 years. It is evident, Franco points out, that heart disease affects workers in their most productive years, when their skill and experience are of the greatest use to industry.

The Consolidated Edison experience is not unique, but is broadly similar to other surveys that have been carried out. Thus, a study by the Bureau of Labor Statistics of the United States Department of Labor may also be cited as typical. Observations were made on 1840 workers with heart disease, who were matched as to age and experience with 3,055 unimpaired workers performing jobs of the same type. The cardiac worker had a slightly higher rate of absenteeism than the control group, but this difference amounted to only two and one-half days more per year. On the other hand, the cardiac group did not display greater proneness to disabling or non-disabling injuries, and what it more significant, they outproduced the healthy controls by an average of 2.4 per cent. In another study in 1944, at the Lockheed Aircraft Cor-

poration, Poole and Bent⁷ found that the claims of 777 cardiacs for lost time payment, hospitalization, medical and surgical benefits were fewer, on the average, than those of the general plant population.

REHABILITATION AND JOB PLACEMENT

With these random comments on the prevalence of heart disease and on the performance record of cardiacs in industry as a background for our discussion, and granted that heart disease is immensely prevalent and that cardiac workers perform well in industry, why, then, is there a problem? There is but one problem in the matter of heart disease in industry but there are many, many facets. As stated by Whitehouse,⁸ at the first meeting of the Rehabilitation Committee of the American Heart Association, "The nature of our problems range from proper education of the young cardiac to Workmen's Compensation, from services at the bedside by the private physician and nurse to industrial medicine, from occupational therapy in the home to sheltered workshop, the Rehabilitation Center, the Custodial Home." Whitehouse personalizes some of the problems by individual comments he has encountered, such as the industrialist who said, "I don't believe in a heart association. Why can't you let the Lord take those he wants?" The union man who said, "I don't care, we're out for all we can get." The physician who said, "It's creeping socialism. I can do the best job right in my office." The cardiac who said, "Let's face it, I have nothing left. I might as well quit and let my kids support me. Nobody wants a guy with a bum heart."

Let us begin with the physician. As far back as 1913, Sir James McKenzie⁹ wrote in his classic book, "A serious responsibility is thrown upon every practitioner, at times, in advising upon certain questions. Should a man give up his business? This is a question in which advice is constantly sought and whether the individual be a statesman or a laborer, the greatest care is necessary in formulating an answer." Even before this remark of McKenzie's, the story of the cardiac in in-

dustry began in this country in New York in 1911, when Dr. Hubert V. Guile organized an evening cardiac clinic for working adults at the Bellevue Hospital. This ultimately developed into the New York Heart Association and, as Kossmann¹⁰ has pointed out, the whole cardiac clinic movement arose from the endeavors to make the indigent cardiac patient economically independent by keeping him on the job. Meanwhile, in other centers throughout the United States, the problem of heart disease in industry was receiving wide attention. Many investigators addressed themselves to the question of heart disease in industry, particularly between 1921 and 1924. In 1935, an important contribution was made by Stroud,¹¹ who published a report dealing with the rehabilitation and placement in industry of those handicapped with the cardiovascular disease.

In 1941, an important forward step was taken at the Bellevue Hospital, where the first cardiac clinic had been organized some 30 years earlier, by the organization of a Work Classification Unit, which has since become a model for many other such units throughout the United States. An important contribution to the problem of heart disease in industry was made in 1952, when members of this group reported their comparison of working and nonworking cardiac patients.¹² In brief, Goldwater, Bronstein, and Kresky found that in a group of 580 cardiac clinic patients whose cardiac lesions had been present for from 5 to 15 years, 81 per cent had continued at work. They made the further observation that there was no evidence that continued employment has any adverse effect on the course of heart disease. A similar study by the Work Classification Unit of the Cleveland Area Heart Society¹³ has yielded similar findings. In brief, in this study comprising 350 patients observed over a period of 2 years, it was found that cardiacs who are not too severely disabled to return to work and who do so with a positive motivation are more likely to improve than to deteriorate, provided their work is adjusted within the limits of their physical capacity. Over the period studied, 23.6 per cent of this group

showed an improvement in cardiac status, contrast to deterioration in 11.7 per cent. A total of 88.3 per cent either improved or remained the same during the period in which they performed work.

These observations that not only can the cardiac work in the vast majority of instances but that his clinical condition may actually be improved by working have been given strong support by recent studies in England conducted by Morris and his associates.¹⁴ These observations have received wide attention in the United States recently, and I need not describe them in detail. Conducted by the Social Medical Research Unit of the Medical Research Council of Great Britain, they show in effect that middle-aged persons engaged in more strenuous occupations actually fare better, in respect to coronary artery disease, than those in more sedentary types of work. Comparison was made, for example, between bus drivers who sit at their work and bus conductors who move about actively; between sedentary postal workers and mail carriers who are active; between amputees with very limited locomotion and those not so handicapped.

ATTITUDE OF THE PHYSICIAN

It is evident, therefore, that much which has been learned requires modification of the dismal view that the medical profession took concerning patients with heart disease only a few decades ago. There remains considerable ignorance among physicians throughout the United States concerning the activities of the patient with heart disease. It may be stated, without fear of contradiction, that less harm is being done to cardiac patients by misdiagnosis and mismanagement of heart disease than results from fear and alarm, indoctrinated in the patient by the doctor, without

to his family as well. A segment of the problem of heart disease in industry still rests within the medical profession with an urgent need for education.

ATTITUDE OF THE PATIENT

The next facet of the problem of heart disease in industry is the problem of the attitude of the patient. This has been very clearly presented by Jezer and his co-workers in the review of their experience at the Altro Workshop in New York City, a philanthropic manufacturing plant given over to the rehabilitation of patients with heart disease and tuberculosis.¹⁴ In a four-year period, 615 applicants in the age range of 18 to 55 years were studied. Of these, 100 who were self-referred were not working because they believed themselves seriously ill with heart disease. The 615 persons studied by Jezer represent a cross section of totally disabled persons of 18 to 55 years of age, and of lower and lower-middle-class economic levels, who do not work because of heart disease, or because of other disturbances confused with heart disease. His findings in this presumably disabled cardiac group are interesting in that only 25 per cent of them had heart disease serious enough to cause disability. The remainder were able to return to gainful employment with reassurance, education, and job placement. Jezer emphasizes that in fully half of the patients studied there was a significant emotional factor superimposed upon the underlying heart disease, which aggravated the disability or was solely responsible for it. Thus, the attitude of the patient as well as that of the doctor is a part of the problem of the employment of the cardiac

ATTITUDE OF INDUSTRY

The next factor, and the major one, in respect to the employment of the patient with heart disease, is the position taken by industry. Not only is an unreasonable attitude common among physicians and patients, as we have indicated, but this feeling may characterize industry as well. As pointed out by Goldwater,¹⁵ in his discussion of heart disease in relation to employment, employers fear the possibility of property damage, Workmen's Compensation or liability claims, and excessive absenteeism and, not uncommonly, express their attitudes bluntly as follows: "Why

should I hire a sick person, if I can hire a healthy one?" The association of fear with ignorance is as true in the case of heart disease as it is in other disorders. There are many reasons why industry fears the employment of cardiacs. Some of these are unfounded, but there are some legitimate grounds for concern. One line of evidence is the growing use of pre-employment examinations that include the use of special diagnostic instruments such as the electrocardiograph. These examinations disclose a large number of instances of heart disease that pose a problem to the employer and it is understandable that where the labor market is plentiful he prefers to avoid employing people with any physical disability. As an instance of the frequency with which the electrocardiograph alone may turn up indications of heart disease, Kline and Hess¹ reported 209 distinctly abnormal electrocardiograms among 2,000 taken in the course of pre-employment and health surveys in a large industry in the Cleveland area.

Although we know that electrocardiographic abnormalities have little bearing on physical capacity, the employer does not know this, and frequently the medical advisers upon whom he relies may make recommendations against employment. In the field of insurance medicine, where my own activities are chiefly concerned, we have, ourselves, undergone a very profound change in attitude with respect to electrocardiographic abnormalities. For example, bundle branch block, formerly considered an ominous sign foretelling a relatively brief life expectancy, was found, in a study we carried out at the Equitable Life Assurance Society, to be much less serious than as is usually supposed, clinically.¹⁶ As a result of our mortality study, which indicates that people with bundle branch block who have no other major cardiovascular impairments have a life expectancy that is almost normal, we now insure such people. Physicians in industry must become more aware that the life and activity of the cardiac need not be compromised, so that he can more properly advise the industry he serves.

PROBLEMS OF LIABILITY AND COMPENSATION

The real crux of the problem of heart disease in industry is the question of liability and compensation, and here the fears of the employer have a substantial basis. This is now being recognized and some attempt is being made to remedy the situation. We may address ourselves to a brief survey of the problem.

Industry's problem with the cardiac is largely a financial one. Some idea of its scope is indicated by the report of Gafafer¹⁹ that industrial absenteeism as the result of cardiovascular disease in the first half of 1952 was exceeded in incidence only by respiratory diseases. Goldwater and Weiss²⁰ remark that in New York State, Workmen's Compensation payments for heart disease amount to about \$2,000,000 annually and account for nearly 4 per cent of all compensation costs.

What is industry doing in practice about employment of cardiacs? Olshansky and his associates conducted a survey in 1954, in an attempt to gather information on some of the problems of the employment of cardiacs in Massachusetts.²¹ This study covered 100 employers in the greater Boston area, representing 176,000 employees, in various types of business and in companies ranging in size from 100 to 20,000 employees. Of the 100 companies, 25 had a stated policy of exclusion of all cardiacs, but, actually, only 51 per cent of the companies were found to hire cardiacs in actual practice. A study of the factors determining the hiring policy regarding cardiacs disclosed that 45 per cent of the firms gave Workmen's Compensation as their first or second motive, followed next by the effect on health and sickness insurance liability, then low productivity, absenteeism, and limited in-plant mobility, in equal proportions. Although Workmen's Compensation claims were not of any great magnitude, it was the sense of threat of cardiac claims under Workmen's Compensation rather than actual experience with them that was the greatest deterrent to hiring of cardiac workers. A similar study was conducted by

Kline,²² who obtained responses to questionnaires from 100 medical directors of industrial organizations throughout the United States, representing some 20 states, and companies that employed 1,340,000 workers. A summary indicates that only 10 per cent never employed cardiacs. Half of the employers occasionally employed cardiacs and the other 23 per cent regularly engaged cardiacs without prejudice. These figures pertain to new employment. It is of interest that 88 per cent of the 100 medical directors reported that their companies always retained workers with heart disease that appeared after employment. As in the study carried out in Massachusetts,²¹ the reason for failure to employ cardiacs was not that they are poor or unsafe workers. Thirty-four per cent of those responding to Kline's questionnaire explained their unwillingness or hesitancy as being due to the threat of monetary loss arising out of compensation claims.

What are the facts of compensation insurance in regard to heart disease? State simply, they are as follows. Unwise legislation, enacted without the benefit of sound medical knowledge, has resulted in a disservice to industry and employees alike. Physicians, cardiac patients, and industrialists have all become victims of a situation not of their own creation. Seriously enough, the pattern has been the same in various states throughout the country. A few illustrations will indicate what has happened. Dr Richard J. Clark,²³ Chairman of the Sub-Committee on Workmen's Compensation of the American Heart Association, reports that in the past few years Board and Court decisions in Massachusetts have so liberalized the interpretation of the law that practically all cardiacs developing acute episodes or dying while on the job are assured of being awarded compensation. It has been stated by the Supreme Court of Massachusetts that occupational strain giving rise to injury need not be unusual or the result of heavy work, and it has also been stated that the stress of strain brought on by worry, fear, or anxiety does not differ from that of physical exertion. It has, furthermore, been ruled that emotional

factors need not be proved by direct evidence, but may be established by reasonable inference. In other words in Massachusetts the floodgates are open wide for decisions in favor of the worker. Under these circumstances, says Dr. Clark, and in view of our knowledge that at least half of all men over 45 years of age have coronary disease, can we justly blame the employer who says he will have no part in hiring cardiacs or people in the older age group? Commenting on the situation in Massachusetts, Sprague²⁴ states that the decision of the Supreme Court of Massachusetts in 1951 has resulted, essentially, in closing the door of employment in that State to anyone known to have heart disease, no matter how minor, no matter what type.

Stout²⁵ has commented on another Supreme Court Decision, in this case in the State of Washington, which has set a precedent in that area. A few years ago, a night watchman for a company in Seattle had a heart attack while sitting at his desk, and died shortly thereafter. The majority opinion of the Supreme Court of the State of Washington held that "the heart attack was due to the strain of the job—that no matter how small the strain, if it were too much for the patient in his existing condition at the time, it was compensable." In New York State, the situation is not far different. Goldwater and Weiss²⁶ report that in 80 per cent of 104 cases before the Workmen's Compensation Board in New York, the onset of symptoms was stated to have occurred while the individual was at work. Among the controverted cases, claims were allowed in 75 per cent of those in which the onset occurred while the claimant was at work. Forty per cent of those had knowledge of pre-existing heart disease. Although a second injury law was enacted in 1945, with the purpose of encouraging employers to hire persons previously partially disabled by placing only limited responsibility on the employer, if further injury or disability occurred while at work, in respect to heart disease, at least, the second injury law has proved inadequate. In the cases studied by Goldwater and Weiss,

although more than 40 per cent had knowledge of pre-existing heart disease, the second injury law was invoked in only one case. Goldwater and Weiss conclude that employers in New York State and elsewhere are becoming increasingly reluctant to hire cardiacs partly because of the manner in which compensation claims for heart disease are adjudicated. In California, similarly as a result of an Appellate Court decision, a serious problem has arisen. As stated by Howard J. Scott²⁷ of the Los Angeles Bar Association for all practical purposes the requirement that the employment cause the heart injury has been eliminated, and the condition that compensation liability arise out of the employment has been eliminated. The cases are now decided purely upon the basis of a requirement that the injury occur 'in the course of the employment'. This means simply that if a man has the onset of his acute symptoms while he is working, the claim is probably one that will be held to be compensable. Scott believes that the interpretation may be extended so that all that is required is that a man have heart disease and that he have a job. Thus, Scott concludes that a tremendous obstacle to the employment of any person who is known to have heart disease has been raised, and the change in the law has had the effect of nullifying all the efforts that have been made and are still being made to encourage industry to employ such handicapped people. Compensation is the major bugbear. There are others, such as the question of group insurance. Persons who are uninsurable for life insurance automatically become eligible for insurance coverage when they are employed in industries having group insurance plans. Surprisingly, group insurance experience has not been particularly bad, and it is questionable whether any appreciable increased cost in coverage results from the employment of cardiacs.

Progress in the clarification of the various aspects of heart disease in industry and in bringing about sound and just practices must come primarily through education and knowledge. The American Heart Association has assumed a very active role in this func-

tion. Work Classification Units, of which about 34 are now functioning throughout the nation, exemplify what can be done. In addition, many of the affiliates of the American Heart Association have special Cardiac-in-Industry Committees, composed of physicians, personnel men, union representatives, people in employment services, rehabilitation, and other social agencies. The American Heart Association Rehabilitation Committee, as already stated, has a subcommittee on Workmen's Compensation. Its Chairman, Dr. Richard Clark,²⁷ recently presented a survey of the problem of Workmen's Compensation in relation to heart disease before the Congress on Industrial Health in Washington, in January, 1955. It appears worthwhile to quote the resolution passed by this Committee of the American Heart Association: "Be it resolved that this Committee of the American Heart Association go on record as recognizing that certain compensation laws and decisions appear to operate as deterrents to full and proper rehabilitation and employment of cardiac patients and, therefore, urges that appropriate reforms be studied and instituted in this field. Because of the broad implications of this matter in relation to other disabling conditions as well as cardiovascular disease, it is our opinion that overall medical details of such reforms should emanate from the American Medical Association."

This, then, is the magnitude of the problem and, obviously, the problem has not as yet been satisfactorily solved. However, in at least one state a noteworthy beginning has been made. In 1952, the Chairman of the Industrial Commission of the State of Utah, recognizing the chaotic state of affairs in decisions where heart disease was involved, requested assistance from the Utah Heart Association. Together with the Salt Lake City Society of Internal Medicine, a committee of internists was designated to draw up for the Industrial Commission a set of guiding principles or criteria for compensability in heart disease. With these criteria as a working basis, the Commissioner then requested that the Medical Society suggest a

panel of nine experts from which he might select three members in rotation to review each case before a hearing. While the decisions of the panel are advisory, the Commissioner makes it clear that he is inclined to accept the panel's opinion. The net result has been that an orderly and sound scientific basis has been established for the handling of claims. Ivan C. Rutledge, Professor of Administrative Law of the University of Indiana, summarizes the cardinal principles of the Utah Plan as follows:

1. The relationship of industrial effort to heart attack can only be determined by recourse to expert medical opinion. Each heart claim is reviewed and evaluated by a panel of physicians.
2. Medical opinion must utilize the most advanced knowledge in the field of heart disease and must be of high caliber.
3. Medical opinion must be based upon complete data in each case.
4. Medical opinion must be unbiased.
5. Medical opinion is obtained before controversy develops.
6. Medical opinion is given to all parties affected by it, including the workman or his beneficiary, the employer, and the insurance carrier.
7. Medical opinion does not deny due process of law.

The Utah Plan has been widely commended and may serve as a model for other States to explore. Certainly it has some faults which have become clear since its inception.

CONCLUSION

In conclusion, I have attempted to survey the magnitude of the problem of heart disease in industry from its many-sided facets. The problem remains a serious one but the obstacles are not insuperable. If there is a single moral to be drawn, it is that we, as physicians, can no longer confine our affairs to a simple physician-patient relationship, but must take cognizance of and leadership in the

many social issues that arise from problems involving the health of the population

BIBLIOGRAPHY

- WHITE, N K, EDWARDS, J E, and DRY, T. J Relationship of the degree of coronary atherosclerosis with age, in men, *Circulation* 1 645, 1950
- GUBNER, R. Diagnosis of arteriosclerosis including observations on lipid metabolism, and ballistocardiogram, *A. Life Insur M. Dr. America, Tr* (1950) 71 20, 1951
- MASTER, A M, JAFFEE, H L, DACK, S, and GRISHMAN, A. Coronary occlusion, coronary insufficiency, and angina pectoris, clinical and post-mortem study, *Am Heart J* 27 803, 1944
- KLINE, E M Aiding the cardiac in industry, *A M A Arch Indust. Hyg* 3 454, 1951
- FRANCO, S C. The experience in industry report of 896 cardiac employees, *New York J M* 56 1428, 1956
- CRAIN, R B, and MISSAI, M E. The employer with heart disease his management in industry, *J.A.M.A* 110 1, 1938
- POOLE, F E, and BENT, J R Employment of cardiacs, problems encountered, *Indust Med* 13 479, 1944
- WHITENHOUSE, F A *The Rehabilitation Program* Remarks at the First Meeting of the Rehabilitation Committee of the American Heart Association, Sept 20-21, 1954
- MACKENZIE, J. *Diseases of the Heart* 3d ed London, H Frowde, 1913
- KOSSMANN, C E, GOLDWATER, L J, and DE LA CHAPELLE, C E Selective placement of patients with heart disease in competitive employment, *Occup Med* 3 531, 1947
- STROLD, W D. Rehabilitation and placement in industry of those handicapped with cardiovascular disease, *J A M A* 105 1401, 1935
- GOLDWATER, L J, BRONSTEIN, L H, and KRESKY, B Comparison of working and nonworking cardiac patients, *A M A Arch Indust Hyg* 5 485, 1952
- HELLERSTEIN, H K, and GOLDSTON, L. Rehabilitation of patients with heart disease, *Podgrad M* 15 265, 1954
- MORRIS, J N, HEADY, J A, RAFFELL, P A B, ROBERTS, C G, and PARKS, J W Coronary heart disease and physical activity of work, *Lancet* 2 1053 & 1111, 1953
- JEZEF, A and BLACK, B S Workshop experience with the "disabled" cardiac, *Brit J Phys Med* 18 8, 1954
- GOLDWATER, L J Heart disease in relation to employment, *J Nat M A* 45 21, 1953.
- KLINE, E M, and HESS, J H. Electrocardiograph in industrial medicine, report based on 26000 electrocardiograms, *Indust Med* 16 378, 1947
- RODSTEIN, M, GUBNER, R, MILLS, J. P, LOVELL, J F, and UNGERLEIDER, H E A mortality study in bundle branch block, *A M A Arch Int Med* 87 663, 1951
- GAFARER, W M Industrial sickness absenteeism rates for specific causes in 1952 for year and last 2 quarters, *U S Pub Health Rep* 68 1052, 1953
- GOLDWATER, L J, and WEISS, N M Study of workmen's compensation and heart disease in New York City, *New York J M* 51 2754, 1951
- OLSHANSKY, S, FRIEDLAND, S, CLARK, R J, and SPRAGUE, H B Survey of employment policies as related to cardiac patients in greater Boston, *New England J M* 253 506, 1955
- KLINE, E M Heart disease and employment—a current survey, *Indust Med* 23 126, 1954
- CLARK, R J, TOUSANT, E S, and SPRAGUE, H B Heart disease in Massachusetts in relation to Workmen's Compensation Act, *New England J M* 252 478, 1955
- SPRAGUE, H B *Personal communication*
- STOUT, C F Industry and cardiac problem etiology of coronary heart disease, *Indust. Med* 23 448, 1954
- SCOTT, H J Employment problems of cardiac, *Indust Med* 23 449, 1954.
- CLARK, R. J *Workmen's Compensation in Relation to Heart Disease* Presentation to Congress on Industrial Health, Washington, D C, January 25, 1955

CHAPTER 48 CAUSAL RELATIONSHIPS IN HEART DISEASE IN WORKMEN'S COMPENSATION CASES

Meyer Texon

Causal relation between an industrial incident or trauma and heart disease is a key question in Workmen's Compensation cases. It must include reference to many contiguous fields. While mindful of legal, social, and economic implications, physicians should consider their proper province to be the medical and scientific aspect of this problem.

Causal relation between trauma and heart disease is part of the study of the etiology of heart disease. A better understanding of this subject requires us to marshal and correlate the pertinent facts established by pathologists, physiologists, physicists, hydraulic engineers, and statisticians, although we may depend primarily upon the accurate and detailed observations of clinicians.

The physician as a scientist must consider and appraise all the facts in a given case. In arriving at his diagnosis or opinion, he must state his conclusions as scientific truth without equivocation and without regard to any social, economic, or legal consequences. Scien-

tific truth does not always provide scientific proof but science may well form a chain of logic leading from fact to opinion so tight and so strong that no speculation can be inserted. Scientific proof does not imply that final answers are available for all medical problems that arise in court. There is always room for honest differences of opinion. The outer limits of justifiable differences are eventually reached. We cannot accept as a certainty what medical science knows to be conjectural and as yet without adequate experimental or clinical proof. All that can be expected is the benefit of the best evidence that science can supply at the present time.

Under current law in most states, compensability for heart disease depends upon the establishment of causal relationship between an occupational incident and a subsequent disability. Some states have Presumptive Acts. The California Presumptive Act rules that heart disease in any fireman or policeman is to be considered of industrial origin unless

unequivocally proved otherwise. New York State has legally defined an accident to have occurred within the meaning of the Workmen's Compensation Law, if it is so interpreted by the "common sense of the average man." It may be emphasized here that scientific facts are established not by definition or legal authority but by experiment. The law may define or interpret an illness or accident as it sees fit but the law cannot alter scientific facts regarding the cause of illness or disease. The physician is not responsible for making such laws; he merely deals with the medical aspects of the problem. When social forces alter the interpretation of the law, the legislators should amend the law without compromising scientific truth and fact.

DEFINITIONS OF TRAUMA

Trauma in its usual sense means injury or wound due to physical force. This concept ranges from the most trivial effect of a change in the environment to the disastrous effect of the H-bomb. In its widest sense the concept of trauma may range from an injury due to emotional factors without physical contact to the effects of the most violent penetrating or nonpenetrating blunt forces. It may also include stress, strain, and effort. These may be physical or mental, usual or unusual, severe or mild, and of either long or short duration.

The question of precipitation or aggravation of an altered cardiac status by the traumatic experience must also be considered. This requires the physician to be familiar with the prior medical status of the heart and all the pertinent circumstances of the injury. The physician must also have a thorough knowledge of the history and natural course of all the various forms of heart disease. Only then can he identify as precisely as possible what agent, disease, or circumstance could be held responsible in a causative sense for the injury or altered status found in the heart. It may require a long experience and a judicial mind to evaluate and allocate the degree of illness due to a disease process and the added disability attributable to an injury.

TYPES OF HEART DISEASE IN WORKMEN'S COMPENSATION CASES

The two cardiac problems most frequently encountered in Workmen's Compensation cases concern the effect of nonpenetrating, blunt force injury to the chest and the effect of effort on coronary atherosclerosis. These two categories comprise about 90 per cent of cases and may be the subject of diverse medical opinion. The remaining 10 per cent of cases involve other kinds of trauma and other kinds of heart disease. Medically, they are generally less controversial.

Penetrating wounds of the heart by gunshot, stabbing, or impalement do not ordinarily present grounds for medical disagreement regarding causal relation between the accident and death or disability. A fatality due to electric shock of the heart cannot be denied. In such a case causal relation is obvious. If the patient survives the immediate effects of electric shock, the residual cardiac disability is usually due to the *status quo ante*, its progressive natural course, and the emotional sequelae. When pulmonary disease of industrial origin causes cor pulmonale, causal relation is clear to all. Trauma and strain do not ordinarily cause rheumatic heart disease. However, the natural course of rheumatic heart disease may be adversely affected by severe strain when the heart disease is considerable.

Congestive heart failure may be induced by severe or prolonged exertion in the presence of heart disease. Such an episode of congestive heart failure may be considered an aggravation or precipitation of heart failure that might occur also as the expected common and natural sequela in any case of advanced heart disease. Thus, strenuous effort may be causally related to acute congestive heart failure in any type of heart disease, such as congenital heart disease, rheumatic heart disease, syphilitic heart disease, hypertensive heart disease, pulmonary heart disease, or arteriosclerotic heart disease, etc. Such an episode of acute congestive heart failure may quickly be fatal owing to the attendant massive pulmonary edema, cardiac

arrest, or ventricular fibrillation. In some cases the fatality is ascribed to the physiological effects of acute myocardial insufficiency, since acute organic changes are absent or appear too mild to account for the death. If recovery occurs, the question of residual disability may, in some cases, be resolved only by accurate observation and evaluation of the subsequent clinical course of the pre-existing heart disease. In other cases it cannot be resolved.

Nonpenetrating Blunt Force Trauma to the Chest

This subject is a controversial one. Each case requires a detailed analysis of the history, physical findings, laboratory data, clinical course and, when available, the necropsy findings. Evaluation of all the facts can usually allocate the degree of damage due to pre-existing heart disease and the added disorder of the heart attributable to injury to the chest.

The earlier literature on this subject is replete with cases of alleged trauma of the heart owing to blunt force chest injury. Many of these are so poorly documented that a scientific appraisal is not possible. The more recent literature on this subject reveals general agreement regarding the exceedingly remote probability of a blunt force injury to the chest causing coronary occlusion with myocardial infarction as an isolated finding. There is also growing agreement regarding the rarity of isolated trauma to the myocardium following nonpenetrating blunt force injury. Most cardiac injuries are associated with other severe chest and body injuries and are immediately or rapidly fatal. Blunt forces severe enough to crush the chest produce not only cardiac but other intrathoracic injuries. These may be found in vehicular accidents and after falls from a height. Contusion of the auricles or ventricles and injuries to the coronary arteries are rarely encountered and never occur as isolated findings. The precipitation of coronary occlusion following a nonpenetrating chest injury depends on the rare instance in which the coronary artery itself is directly involved. This most unusual cir-

cumstance cannot be called upon to explain the numerous cardiac symptoms that may follow nonpenetrating chest trauma.

Arteriosclerotic Heart Disease—Coronary Occlusive Disease

The natural history of arteriosclerotic heart disease in the working population is the same as it is in the total population. It is not surprising, therefore, to find that the major problem of heart disease as encountered in Workmen's Compensation claims is concerned with arteriosclerotic heart disease in all its varied forms. In a recent study of 100 consecutive Workmen's Compensation cases involving heart disease there were 78 instances in which significant coronary atherosclerosis was diagnosed.¹ In 11 other cases coronary atherosclerosis was considered relatively asymptomatic, the principal etiologic factor being hypertension, rheumatic fever, syphilis, or pulmonary disease. In the remaining 11 cases, no significant organic heart disease at all was diagnosed.

Symptoms indicative of coronary occlusive disease appear when the coronary blood flow is impaired or is insufficient for myocardial demand. The evolution of coronary atherosclerosis may produce a variable clinical picture owing to a variety of pathologic changes leading to diminution of the caliber of the lumen. The clinical picture may represent a physiologic response and in many cases cannot be correlated with the pathologic findings. A report discussing the correlation of clinical and pathological findings in these cases is being presented elsewhere at this meeting.²

The diminution of the caliber of a coronary artery is usually the result of slowly progressive coronary occlusive disease with thickening of the intima. The flow of blood may be altered more abruptly by thrombosis at the site of atherosclerosis or by hemorrhage into an atherosclerotic plaque. These events, however dramatic or even fatal, can be shown to be complications in the evolution of atherosclerosis. We cannot proceed to the question of causal relation without presenting at this time recent findings that shed light on

the localization and inherent pathology of atherosclerosis. A recent study appears to demonstrate the primary relation of hemodynamics to the atherosclerotic process.²

Hemodynamics. The Primary Factor in Atherosclerosis

The laws of hydrodynamics apply to the circulatory system. Hemodynamic factors can account for the localization and progressive pathologic development of arterial occlusive disease in the coronary arteries as well as in the aorta or other arteries.

The distribution of atherosclerotic lesions indicates that certain points of predilection are determined by the nature of the motion of the blood. Arterial blood flow is generally streamline or laminar in character (1) In such tubes with converging boundaries, velocity of flow increases and static pressure decreases in accordance with Bernoulli's Law (2) In curvilinear motion, static pressure increases along the outside curvature owing to centrifugal force while there is a decrease in lateral pressure on the inner curvature that is directly proportional to the same force (3) Since the velocity of flow is greatest in the axial line, the impingement of the more central blood stream upon a site of branching or bifurcation causes an increase in velocity pressure and decrease in static pressure at the medial walls of the crotch zone

These physical factors clarify the predilection of atherosclerotic lesions for areas in the circulation characterized by converging boundaries, branching, bifurcation, abrupt curves, and points of external attachment. At these points the blood flow is subject to an increase in velocity and to a decrease in static pressure. The local decrease in static pressure may be considered a suction effect that produces the initial tendency for the vessel to be narrowed by movement of the wall toward the center of the lumen. This gives rise to loosening of the intimal stroma and the initial reaction of intimal thickening by raising the cells of the intimal surface. At such areas of intimal change, continued exposure to the suction action of the blood stream stimulates further changes. These lead

to the formation of the characteristic atherosclerotic plaque. This process involves lining endothelium, subendothelial fibrous layer proliferation, variable lipid accumulation, cellular infiltration, fibrosis, and frequent calcification. Capillary vascularization from the lumen and the vaso vasorum may occur. The atherosclerotic plaque is subject to motion, owing to the varying suction effect that is dependent upon the velocity of blood flow at the area of narrowing and not necessarily directly related to the systemic blood pressure. When the pathologic degenerative changes in the atherosclerotic plaque have reached a critical point, the usual velocity and lateral pressures or a local increase in velocity of flow with corresponding decrease in static pressure may cause detachment of the intimal layer. This produces a raw or ulcerated atheromatous surface which, in contact with the flowing blood, may result in the formation of a mural thrombus at this site.

Thus, the composite effects of blood flow, external attachment, caliber of the lumen, degree of curvature, and branching characteristics determine the sites of predilection and pathogenesis of atherosclerosis as well as its complications. It appears that the behavior of fluids in accordance with the laws of fluid dynamics accounts for the location of an atherosclerotic plaque and its progressive pathologic evolution.

Varying types and degrees of occlusion develop as a result of variations in the pathogenesis (1) Coronary occlusion may result solely from progressive intimal thickening or atheromatosis. Pathologic examinations of such occluded coronary arteries will disclose that the atheromatous process developed at a site of predilection influenced by increased blood velocity, impingement of blood stream, attachment, or curvature. A relatively firm and straight vessel may show concentric or more concentric atherosclerosis. The nature of flow in a zone of sharp curvature determines the greater degree of thickening of the internal radius in comparison with the external radius. The free peripheral arc of the involved coronary artery may be less affected than the attached myocardial arc. The

duced lumen at the site of such atherosclerotic change will be, of necessity, eccentrically placed (2) The local increase in blood velocity and decrease in static pressure at the site of an atherosclerotic plaque cause movement of the vessel wall with a tendency for the plaque to be drawn toward the center of the lumen This mechanism may account for hemorrhage in the adventitia, media, or intima of atherosclerotic vessels Intramural hemorrhages occur most frequently in association with atherosclerotic plaques The occurrence of a subintimal hemorrhage in an atheromatous plaque is a complication that may contribute further to the occlusive changes (3) The increased blood velocity and decreased static pressure at the site of a soft atherosclerotic plaque may shear off and embolize the superficial intimal layer or the contents of a plaque This exposes a raw, irregular atherosclerotic ulcer surface to which blood elements become attached to form a thrombus, which may then become occlusive.

The flow of blood in the coronary arteries is intermittent as a result of systolic contraction The stream is subject to rapid changes in velocity, increased flow occurring during diastole, and markedly reduced flow during systole The coronary arteries appear to be unique in the body with respect to such phasic variations in blood flow It is also notable that the caliber of the extramural coronary arteries tapers rapidly These may be significant factors in their predisposition toward atherosclerotic changes

It appears from the above-described hemodynamic mechanisms that occlusive changes in the coronary arteries are the sequelae of fluid dynamics applied to the natural conditions of the coronary circulation

Relation to Stress, Emotion, and Effort

The velocity and volume of blood flow in the coronary arteries are increased when the body requires the heart to increase its rate With increasing tachycardia, the volume and velocity of coronary flow eventually diminish owing to the shortened diastole

Similarly, a strict correlation between heart

rate and cardiac output does not take place, the output rising with increasing heart rate until a critical point is reached at which the cardiac output is reduced by the small stroke volume. If the velocity of coronary blood flow is increased by episodes of exertion, emotion, or stress, then the possibility exists that a previously existing atherosclerotic plaque could undergo anatomic change as a result of the decrease in static pressure at this site. This effect would be immediate with respect to intramural hemorrhage or ulceration of an atherosclerotic plaque with thrombus formation. From the standpoint of clinical correlation this could be the mechanism of some sudden deaths resulting from acute coronary occlusive disease with antecedent progressive coronary atherosclerosis This may be the mechanism in the few cases of acute coronary occlusion with myocardial infarction that appear to follow upon very strenuous exertion or extreme emotional disturbances It should be emphasized that sudden death can occur from coronary insufficiency incident to exertion or emotion without sufficient time for myocardial infarction to result Coronary insufficiency may precipitate a fatal arrhythmia such as cardiac standstill or ventricular fibrillation Also, coronary insufficiency may progress to myocardial infarction without any additional acute occlusive process An exertional or emotional episode that produces hemodynamic alterations sufficient to cause acute occlusive complications in the pre-existing sclerotic disease of the arteries may result in bridging symptoms between the episode and the subsequent development of myocardial infarction or death In such a case, the causal relation appears plausible and cannot be excluded. If an interval elapses without significant symptomatology, the causal relation would be speculative. In the absence of bridging symptoms there does not seem to be sufficient reason to relate myocardial infarction or death due to occlusive coronary artery disease, with or without acute luminal thrombosis or intramural hemorrhage, to a previous episode of stress that may have produced a transient increase in velocity or decrease in static pressure of

coronary blood flow. The natural course of occlusive coronary artery disease, influenced by the usual wide range of hemodynamic changes of velocity and lateral pressures in the coronary circulation, can account for all but the most exceptional cases of myocardial infarction or death resulting from coronary occlusive disease.

SUMMARY

The question of causal relation in Workmen's Compensation cases involving heart disease requires the correlation and evaluation of scientific facts established by physiologists, pathologists, physicists, hydraulic engineers, statisticians, and clinicians.

It is necessary to know the natural history of all forms of heart disease before attributing an altered cardiac status to any traumatic experience.

Nonpenetrating blunt force injuries to the chest rarely, if ever, cause coronary occlusion with myocardial infarction as an isolated finding. Myocardial contusion or laceration, similarly, does not ordinarily occur as a result of chest trauma unless the injury is severe enough to crush the chest and produce severe associated injuries to the chest wall, ribs, and intrathoracic organs.

Emotional sequelae of traumatic experiences can simulate organic heart disease symptomatically.

Congestive heart failure with subsequent diminution of cardiac reserve can be precipitated by severe exertion in the presence of any form of moderate or advanced heart disease.

Arteriosclerotic heart disease in all its

varied forms accounts for about 90 per cent of all Workmen's Compensation cases involving heart disease.

The causal relation of stress, effort, and emotion to coronary occlusive disease is discussed. The central role of hydraulic forces applied to the coronary circulation is described. The hemodynamic factor is presented as the primary cause of atherosclerosis. The question of causal relation between a traumatic experience and an acute episode in the course of coronary occlusive disease is discussed from the standpoint of hemodynamic and clinicopathologic correlations. It appears that the natural course of coronary occlusive disease due to atherosclerosis is primarily influenced by the local hemodynamics. The usual local hemodynamics can account for all but the most exceptional cases of coronary occlusive disease resulting in myocardial infarction or death.

BIBLIOGRAPHY

1. TEXON, M. *Heart Disease and Industry, with Particular Reference to Workmen's Compensation Cases*. Forewords by S. A. LEVINE and H. W. SMITH. New York, Grune & Stratton, 1954.
2. WEINBERG, S. A., and REIFERN, M. Sudden death and circumstances in coronary artery and other types of heart disease. First Wisconsin Conference on Work and the Heart, New York, Paul B. Hoeber, Inc., 1959, Chap. 43, p. 288.
3. TEXON, M. Hemodynamic concept of atherosclerosis with particular reference to coronary occlusion, *A. M. A. Arch. Int. Med.* 99:418, 1957.

CHAPTER 49 EMPLOYMENT OF THE CARDIAC

Irvin Klein

Until recently, the skills of physicians were directed primarily to the treatment and cure of physical disease. Mental and psychic illness was almost entirely neglected. The rehabilitation of those who had been injured or were sick was given little thought. The effects of the illness on the patient's industrial capacity and future livelihood, on his morale and on his social and familial welfare were little considered.

The loss or diminution of acquired skills as a result of injury or disease deprives the patient of an essential prerequisite for earning his living, and for maintaining his physical and mental well-being. It deprives industry and society, also, of the fruits of these skills. For industry suffers more through the impairment of a worker's health and the consequent loss of his talents than through the breakdown of a costly machine. The latter can easily be replaced. The former cannot.

To overcome the unnecessary diminution of these special skills is one of the aims of rehabilitation medicine, of vocational guid-

ance, and of selective placement. Relatively few patients are permanently, totally disabled. The great majority have only a partial disability, either temporary or permanent. It is essential, therefore, that the patient's attention be focused on his residual capabilities rather than on his irreparable disability, and that these residual capabilities be employed and be further developed. Through rehabilitation an attempt is made to restore, as far as is possible, the maximum physical, mental, and vocational capacities to a handicapped patient.

In order to achieve this end, it is necessary to have the thoughtful and willing co-operation of the general practitioner with experts in diverse fields—medicine, psychiatry, surgery, social service—as well as the assistance of those qualified in the retraining of the handicapped, of employers, and of governmental agencies.

The general practitioner is of especial importance. It is he who examines applicants for work in the smaller industrial plants and who decides on whether to accept or reject

the applicant. It is he who must learn that physical perfection is not a *sine qua non* of employment, and that a rehabilitated, partially disabled workman can and often does out-perform his physical betters. It is the general practitioner who, together with the employment specialist of small industries, decides on the working fate of the sickly or injured. It is he who usually rejects them, for fear that a second injury may be too costly in compensation benefits.

"Each of us," it has been said, "is an unpregnable fortress, which can be laid waste only from within." Unemployment and idleness—especially unnecessarily enforced idleness—are among the more important destructive factors. They produce personal debasement owing to the resultant worry, want, and privation. Self-esteem is lost too soon, morale broken too often, and family ties snapped too frequently. Unemployment must, therefore, be avoided whenever possible. But a handicapped person's employability depends not only on his own willingness to work within his physical capabilities, it depends in great part, as well, on the willingness of smaller industrial plants to employ him, since the majority of the workers in the entire labor pool are used in small industry.

The employee's willingness to work is affected in great part by his physician and his family. Commonly, as in cardiac disease, the physical disability is not of overwhelming significance. But the fear of heart disease and of its dread consequences, thoughtlessly inspired by the attending physician and carefully nurtured by the patient, his wife, and his children, is the important cause of the proclaimed disability and of the unnecessarily enforced idleness.

An effort must be made to raise the spirit of hope from the deep valley of despondency. Attention should never, therefore, be focused solely on the heart condition. The physician must consider the patient and should not treat only the disease. Since most workmen can understand simply expressed facts concerning their disease and its outlook, these facts should be clearly stated, as soon as it

seems wise to do so, and in as optimistic a manner as is commensurate with the existent condition. A closer view of any problem and a greater understanding of the problem dissipate much of the fear inherent in it.

Complete functional cure of coronary occlusion is a rather common finding. A partial disability usually results, from an incomplete functional cure. Most such patients can return to full- or part-time work especially if it is not of too strenuous a nature, since work only rarely demands the maximum of one's normal functional capacity.

Those who suffer from the intermediate syndrome of coronary insufficiency or from occasional attacks of angina pectoris may resume lighter work. They can be assured that most such attacks can be prevented by a judicious avoidance of the precipitating physical stresses and emotional strains, and by the use of nitroglycerin before and during an attack. The willingness—even the unwise eagerness—of a sufferer from heart disease to work can easily be affected by the sage counsel of his attending physician. Would that it were as easy to induce smaller employers to engage handicapped cardiac patients.

All endeavors that affect the entire community and are subject to public scrutiny must have, as their base, good public relations. A smaller employer can hardly be expected to engage a handicapped cardiac if he has little faith in the statements that the physician makes, and if the monetary compensation risk he is asked to take is beyond reason. The employer must be impressed with the honor, the ability, and the impartiality of the physician. He must be wholly imbued with confidence in the physician, and must trustfully accept the medical data presented to him and the medical conclusions based thereon. He must be assured that the medical reports furnished are adequate, in that they adhere to the known facts, and that the medical testimony given is beyond question, in that it expresses the views held by a majority of authoritative cardiologists.

To attain this end is difficult. Individual general practitioners, striving alone, will rarely be successful. They must seek the help

and guidance of local heart chapters and the local medical groups, who must present the medical and social problem in its best possible light, from the view of both the patient and the employer. Together, they must publicize the problem of the unemployed cardiac, they must dwell upon the benefits that both the employer and employee will reap from a solution of the problem, they must attract the co-operative efforts of government, labor, industry, and the medical profession, and they must persuade the entire community to have confidence in their good faith and good judgment.

The usefulness of the disabled cardiac in industry has already been sufficiently established by experience. Each sufferer from heart disease who has been permitted to resume work has evinced an ability to do his assigned task, and has shown a devotion to duty that compares very favorably with that of his healthy co-employees. Absenteeism among employed disabled cardiacs is relatively low, as it is among all rehabilitated partially disabled workmen. Industry thus also gains through giving jobs to those who are handicapped.

Experience has also taught, however, that employment of disabled cardiacs should not be a chance, haphazard matter. The job must suit the particular employee and each employee must suit the particular job to which he is assigned.

Occupation, like education, should tend to promote a happily sufficient life for each individual. Vocational guidance, which is an essential component of rehabilitation, seeks to find for each disabled person not only the job for which he is best qualified physically, but also the job in which he will be most contented.

The great forces that the larger industries can muster must here come into full play. All available jobs must be carefully studied and classified. This does not mean a mere listing of the various jobs under their various titles, for this is worthless when a disabled cardiac is to be assigned to work. It does mean a carefully detailed description of the work en-

tailed in each job and the duties that devolve upon the prospective employee.

The cataloguing of the different jobs at each large industrial plant must be under the concerted supervision of the personnel officer, the immediate foreman, and the plant physician.

As industry must scrupulously determine the exact physical demands that each job makes, so must medicine and its allied arts scrupulously determine not only the exact physical abilities of the handicapped, but also, if possible, the psychoemotional effects that a contemplated job may have upon him. This is probably among the most difficult tasks in medicine, but if successful it is also the most rewarding, for upon the conscientious accomplishment of this task rests the entire program of re-employment of the cardiac. Its successful achievement must depend on the close co-operation of the attending physician, the cardiologist, the plant physician, social service agencies, sheltered workshop directors, work classification units, and other localized community enterprises. Chiefly, of course, will it depend upon the active, voluntary co-operation of the patient, and on his entire attitude toward resuming work.

The physical ability of a cardiac patient can be determined by various criteria: the physical examination, the frequency and severity of attacks of pain, the degree of effort that results in pain and dyspnea, and the results of tests of cardiac function, if their performance is not contraindicated. An exact physical diagnosis is essential. Vague statements concerning the presence of a "weak heart" or a "faint murmur" are not only valueless but may even be harmful. They may unnecessarily limit the activities of an individual whose heart is normal functionally, or may needlessly permit a cardiac to work at a job for which he is wholly unqualified.

The re-employment of a cardiac, however, and the vocational guidance that leads to his rehabilitation, are dependent not only on his physical capability. In many cases, a complete retraining of the individual becomes

TABLE 49-1. HEART CASES UNDER THE SECOND INJURY LAWS*

<i>I Includable as "Prior Injury"</i>	
California	Washington
District of Columbia	West Virginia
Florida	Wisconsin (same as I)
Kentucky (although "disease" excluded, unless from injury, or from service in armed forces)	Wyoming (must be "subsequent compensable injury")
Minnesota (although doubtful as to "degenerative diseases," as in <i>Sensle</i> case)	<i>III Includable as Both Prior and Second Injury</i>
Missouri	California
Nebraska (although prior disability due to "Disease" excluded)	District of Columbia
New Jersey	Florida
New York	Kentucky (see I)
North Dakota (although must have been caused by employment)	Missouri
Ohio (wherein cardiac disease and cerebral vascular accident are specifically included)	Nebraska (see I)
Oklahoma (includes "any disability," if previously so determined by the Commission)	New Jersey
Rhode Island (although progressive deteriorative conditions or diseases are ruled out)	New York
Utah	North Dakota (see I)
Washington	Ohio (see I)
West Virginia (although must be a "definitely ascertainable impairment" caused by a previous injury)	Oklahoma (see I)
Wisconsin (although limited to a permanent disability which alone would have rated 250 weeks' benefits, less 2½ per cent each year over age 50, up to 50 per cent deduction)	Utah
<i>II Includable as "Second Injury"</i>	Washington
California	West Virginia (see I)
District of Columbia	Wisconsin (see I)
Florida	<i>IV Includable as Either Prior or Second Injury (or both) but Where Permanent Total Disability Is Required for Contribution by Fund</i>
Kentucky	District of Columbia
Missouri	Minnesota
Nebraska	Nebraska
New Hampshire (must be "further permanent disability")	New Jersey
New Jersey	Rhode Island
New York	Washington
North Dakota	West Virginia
Ohio	<i>V Includable as Both Prior and Second Injury and Where Permanent Total Disability Is Not Required for Contribution by Fund</i>
Oklahoma	California
Utah	Florida
	Kentucky
	Missouri
	New York
	North Dakota
	Ohio
	Oklahoma
	Utah
	Wisconsin

CONCLUSIONS

1. There are 17 states (including the District of Columbia) in which a heart injury may conceivably be considered a "prior injury" or "impairment." It is important to note, however, that in eight of these there are limitations placed, either by statute or

* Compiled for the American Heart Association

TABLE 49-1 HEART CASES UNDER THE SECOND INJURY LAWS (Continued)

case law, upon the definition of "prior injury" which may, therefore, exclude heart injuries *

2. There are 17 states (including the District of Columbia) in which a heart injury may conceivably be considered a "second injury" **

3. There are 15 states (including the District of Columbia) in which a heart injury may conceivably be considered as both a "prior injury" and a "second injury" †

4. There are only ten states where a heart

injury may conceivably be considered as both a "prior injury" and a "second injury," where the combined disability need not be permanent total for the second injury fund to come into operation. These ten states, as listed in V, above, are to be considered the most liberal or broadest in their possible application to heart cases, from the point of view both of contribution by second injury funds and of aiding the cardiac who is not permanently and totally impaired and may be a candidate for rehabilitation.

* NOTE The 17 states mentioned in Conclusions 1 and 2 are not the same in all cases. Of those 17 listed in I, above, Minnesota and Rhode Island are not included in II, above. Of those listed in II, New Hampshire and Wyoming are not included in I. Hence, we arrive at the listing of the 15 states in III, above, as given in Conclusion 3.

imperative. In relatively few cases, when congenital heart disease or early acquired disease had prevented the patient from seeking work previously, education for work is necessary. If the determination of a patient's physical capacity for work is difficult, how much more difficult is it to determine his likes and dislikes, his aptitudes, his psychic reaction to the work contemplated, his personal and familial background, his formal training, and his knowledge. Various psychological and intelligence tests should, of course, be undergone. But the results obtained are too often obscure and not too rarely erroneous. Great care, much competence, and an unusual understanding of the patient and the problem are essential, if the examiner is to reach the correct conclusion in each particular case. Should an incorrect conclusion be drawn, the training to which the patient had been subjected will be in vain; he will be a failure at the work given him, and an entirely new course of training will have to be instituted, with even greater hardships and greater difficulties in readjustment.

Having been carefully studied physically and psychologically, and having been trained for a particular job, which also has been carefully analyzed, the cardiac is now ready for his work. This constitutes selective placement. The employer must then be induced to

engage the services of the trainee at the particular work for which he has been found to be qualified. The employer must be made to understand that job changing is not permitted without the approval of the plant physician, the personnel director, and the foreman—all of whom know the peculiar demands that each job makes. For if a disabled rehabilitated cardiac is compelled to do work beyond his capabilities, the entire selective placement program will of necessity fail. The failure of an individual disabled worker will make more difficult the future placement of other workers. Several such failures may lead the employer to decide never to engage the disabled.

Industry will thus lose much-needed skills, the worker will lose his income and his self-esteem, and society will not only lose a self-sustaining citizen, but will also be compelled to help in the support of him and his family.

In order to foster the employment of the cardiac and to diminish the increased compensation cost to the employer resulting from partially disabled employees, forty-three states have enacted some form of "Second Injury Law." The New York State Law has been outlined briefly recently.¹ It provides that the employer is responsible for only 104 weeks of compensation benefits in second injury cases, certain prerequisites having been fulfilled.

Whether such a diminution in the employer's liability will stimulate his employment of the disabled is not yet clear.

The average time loss of work-connected disability is often much below this figure. If it were possible to hold the employer of the disabled responsible for only 26 or 52 weeks' compensation in second injury cases, an impetus might be given to a more widespread hiring of disabled men and women by industry.

Of the 43 states that have enacted Second Injury Laws, relatively few, unfortunately, make any provision for the re-employment of cardiacs (Table 49-1). This appears to be an unwise omission. The ability of an individual to do some particular work cannot always be determined by his physical status alone. Aptitudes and skills are not peculiar to those who suffer from illnesses or injuries that are especially mentioned in the Law. An alert

medical profession should urge that this legal oversight be corrected.

CONCLUSION

The re-employment of the disabled cardiac, as well as of other disabled workers, is a consummation devoutly to be wished. What rehabilitation has already done is praiseworthy. What vocational guidance and selective placement can accomplish further depends, in great part, on the general practitioner and his willingness to learn a new and valuable method of therapy.

The employment of the partially disabled is a boon to him, to his family, to industry, and to the community as a whole.

BIBLIOGRAPHY

1. KLEIN, I. Employment problems affecting the cardiac, *New York J. Med.* 56:2273, 1956.

CHAPTER 50 WORKMEN'S COMPENSATION—AN INDUSTRIAL AND SOCIAL MECHANISM

Henry D. Sayer

It is appropriate that a panel discussion of *Work and the Heart* should be initiated with a consideration of the entity that we know as *Workmen's Compensation*. To that end, and as a preliminary to a review of the whole subject, it is well to make certain that we understand just what we mean by *workmen's compensation* and the relations it has to the subject matter of our thoughts here today.

We find workmen's compensation being brought into the discussion most often when the impact of heart disease upon industry is being considered. Let us then ask ourselves, "What is workmen's compensation?" To many of us that seems an unnecessary question; but to many others the term is used without a complete understanding of the subject or of the history, background, objectives, and philosophy of this system, which has become a characteristic of the modern industrial life that influences so greatly our economic and social thinking.

Webster defines workmen's compensation act as: "Any of various State statutes fixing

the compensation that a workman may recover from an employer in case of accident (and sometimes of occupational disease) arising out of the employment, giving to a workman, except when an injury is willfully self-inflicted or caused by intoxication, indemnity according to a definite schedule without regard to fault." [Enlarged by permission From WEBSTER'S NEW INTERNATIONAL DICTIONARY, Second Edition, copyright 1934, 1939, 1944, 1950, 1953, 1954, 1957, 1959 by G & C. Merriam Co.]

This definition is provocative of the myriad questions that it suggests but does not fully answer. It is impossible in a brief statement to give a clear idea of what this multistate all-embracing workmen's compensation system is. The law in each state differs from the laws in all the other states in some particulars. They differ in benefits, in coverage, in procedure, in administration, in security provisions, and in many other ways. And yet they all are clearly recognizable as compensation statutes.

HISTORICAL DEVELOPMENT

Accordingly we may well look back over the years in order to attain a perspective of the system of workmen's compensation and employers' liability. In doing so we may note what the employer had to meet in the way of liability to his injured employee and what rights against his employer the injured worker had before the opening of the *compensation era*.

If we go back far enough, we find that the employer owed no specific duty to his employee other than that which he owed to the stranger. This concept began to undergo change in later years, and the employer began to be charged with a special liability to his employee as such. This was not a matter of statute; it was the *common law*. Advances along this line were painfully slow, but the industrial day had scarcely begun to dawn.

The principles of compensation came down from the earliest times, for we find in Exodus, Chapter 21.

Verse 18. And if men strive together, and one smite another with a stone, or with his fist, and he die not, but keepeth his bed

Verse 19. If he rise again, and walk abroad upon his staff, then shall he that smote him be quit: only he shall pay for the loss of his time, and shall cause him to be thoroughly healed.

Thus we see that the basic principles of the compensation law were enunciated by Moses when he came down from Mt. Sinai.

The first laws with which we need concern ourselves were the laws setting up an industrial insurance plan in Germany under Bismarck in the early 1880's. The German system was divided into two major divisions, one related to nonoccupational disability, whether it resulted from an accident or from disease or even just old age, the other related to accidents that were work-related, and for which the employer was charged with certain liability. This was probably the first clearly defined workmen's compensation law and it had an important influence on world thought.

This was at about the time that the English common law was supplemented by the

statutory Employers' Liability Law of 1880. The proof required under this statute, however, was so rigid and its provisions so difficult to meet that relatively few persons availed themselves of it, and the force of public opinion brought about an abandonment of the 1880 law in favor of the English workmen's compensation statute, which was enacted in 1897. The 1897 Workmen's Compensation Act, however, did not inspire great confidence in the public mind. It was a voluntary system, it did not debar workers from suing under the old common law where negligence could be proved and its coverage was narrow and confined to small areas of hazardous employment. A few years later it was superseded and the coverage extended in a new enactment, the Workmen's Compensation Law of 1906.

Our Federal Constitution recognized the common law of England as the law in this country on all matters and questions as to which the Congress or the legislature of the several states had not by statute expressly provided. Accordingly, in the field of employers' liability the English common law prevailed until such time as the states made explicit provisions. In time, such state statutes were generally enacted. These laws, however, proceeded on the general principles of the common law, that is, they all were based on fault or negligence on the part of the employer, though to some extent mollifying the harshness of the common law defenses. Rather generally they gave a right of action in case of death due to accident, whereas the common law of England did not. This was then the situation in the United States prior to 1908. Widespread dissatisfaction was building up in industry and labor because of the inadequacies of employer's liability laws. Relatively few cases succeeded in meeting the requirement of proof of the employer's negligence and the employee's entitlement.

With all the uncertainties, with all the delays of court procedure, with hungry mouths to feed at home and other expenses to be met, settlements under pressure became the rule, settlements often improvident and at

figures far less than were warranted by the circumstances. Even when settled at a fraction of the correct value of the case, deductions had to be made for payments of the lawyer's fee and other expenses, including medical care. When he got his net share of the recovery, the employee sometimes wondered who it was who had been injured.

Is it any wonder, then, that the minds of the people, workers, employers, and others alike, sought for a better, a fairer, a more expeditious way of dealing with the problem of the worker injured or killed at his job? It needed only a leader, a clarion call, to rouse the conscience of America. Such a call was sounded. On January 31, 1908, President Theodore Roosevelt, in a message to Congress, declared it to be a national duty to enact laws that would bring relief to workmen injured in their employments. The authority of Congress to act in such matters was limited under the Constitution to employees of the government itself and employees of interstate carriers engaged in interstate commerce.

The President, in his message, referred to the Employers' Liability Law for railroad workers, and then went on to propose and recommend an act for government workers injured in their work to provide for them and their dependents limited benefits without regard to fault as a cause, and such benefits to be extended in a speedy, summary manner, without technicalities such as prevailed in actions in the courts. He went on to say:

I also very urgently advise that a comprehensive act be passed providing for compensation by the government to all employees injured in the government service. Under the present law an injured workman in the employment of the government has no remedy, and the entire burden of the accident falls on the helpless man, his wife, and his young children. This is an outrage. *It is a matter of humiliation to the nation that there should not be on our statute books provision to meet and partially to atone for cruel misfortune when it comes upon a man through no fault of his own while faithfully serving the public. In no other prominent industrial country in the world could such gross injustice exist.*

MOST ALL civilized nations have enacted legislation embodying the complete recognition of the principle which places the entire trade risk upon industrial accidents (excluding, of course, accidents due to wilful misconduct by the employee on the industry as represented by the employer, which in this case is the government. . . . Exactly as the working man is entitled to his wages, so he should be entitled to indemnity for the injuries sustained in the natural course of his labor.

The rates of compensation and the regulations for its payment should be specified in the law, and the machinery for determining the amount to be paid should in each case be provided in such manner that the employee is properly represented without expense to him. . . .

The same broad principle which should apply to the government should ultimately be made applicable to all private employers. Where the nation has the power it should enact laws to that effect. Where the States alone have the power they should enact the laws.

This was the call that was needed. Congress promptly passed a United States Employees Compensation Act applying solely to government employees, and at the same session re-enacted a Federal Employers' Liability Law applying to employees of railroads injured in work that was in interstate commerce. Following this message from the President, the legislatures and the governors of various states became active in planning laws for their states to provide a system of workmen's compensation for employees subject to state laws. Following the message of the President, commissions were appointed to study the subject in New York, Wisconsin, and Minnesota. At the same time the problem was being studied in other states. In the state of New York, Governor Charles E. Hughes, in a message to the legislature in January, 1909, urged action for a study of the subject as follows:

I also recommend that provision be made for special and expert inquiry into the questions relating to employers' liability and compensation for workmen's injuries. Our present methods are wasteful and result in injustice. . . . There are constitutional restrictions which stand in the way

¹ Special Message, President Theodore Roosevelt, 1908.

of some of the remedies which have been devised in other countries; but the subject should be thoroughly examined to the end that the present want and injustice should be mitigated to the fullest extent that may be found to be at once practicable and consistent with our fundamental law.²

In accordance with the Governor's recommendation, the legislature created a Commission on Employers' Liability Laws, of which Hon. J. Mayhew Wainwright, a state senator, was chairman.³ This Commission reported to the legislature on March 19, 1910, in a voluminous and significant report with statistical tables, reports of various surveys, legal and expert opinions on many subjects related thereto, with the complete testimony. It developed a draft of compulsory workmen's compensation law for certain hazardous employments and a voluntary law for other employments generally.⁴

This New York law thus became the first workmen's compensation law to be enacted in the United States covering private employments. However, this law was declared unconstitutional by the New York Court of Appeals in 1911, in the *Ives* case.⁵ At the earliest date possible thereafter, the Constitution was amended in 1913 by vote of the people to validate a workmen's compensation law. In 1911, several states enacted workmen's compensation laws, among them Wisconsin, New Jersey, Massachusetts, and Washington, and other states soon followed. The revised New York Workmen's Compensation Law became effective on July 1, 1914, and its constitutionality has been sustained by the United States Supreme Court. By the end of five years, at least 26 states had enacted workmen's compensation laws, and within a few years thereafter, all 48 states had such a law. Congress had enacted the Longshoremen's and Harbor Workers' Compensation

Act, and that Act was later extended to all employees of private employers in the District of Columbia. Workmen's Compensation laws have also been enacted by Hawaii, Alaska, and Puerto Rico. There has thus come about an acceptance of the principle of workmen's compensation in every corner of this country and its territorial possessions.

I like to think of workmen's compensation as a system of laws rather than as a particular law. In truth, it is a system. There are in this country⁶ 48 state laws, three federal laws and three territorial laws. All of them are designated by the general term *workmen's compensation law*. Probably no two of these laws are identical in all respects, even though some of them are cut in the same pattern. But as we have said above, they all have that something, that universal quality that entitles them to acceptance as workmen's compensation acts and part of a common system. A principle that seems to me to underlie all compensation laws, without exception, is that *the right to compensation arises without regard to fault as a cause*. That single, simple phrase, implicit and explicit in all compensation laws has, over a period of slightly less than half a century since the introduction of the system in this country, worked a momentous change in our whole industrial, economic, and social life. Once we remove the squabbling element of fault, we find we can approach the problem of the industrial accident in a broader, more comprehensive and more intelligent manner.

PHASES OF WORKMEN'S COMPENSATION

The workmen's compensation system in its entirety, as we view it, divides itself into three distinct phases: first, accident prevention, and in that, of course, we include industrial hygiene; second, curative, which includes not alone medical and surgical care, but must include rehabilitation or restoration; and third, compensatory or relief. I put these phases in what I believe is the order of their importance in our economy.

⁶The conference on *World and the Work* was held in 1927.

²Annual Message of Gov. Charles E. Hughes, N.Y., to legislature, January 1 1909.

³Commission appointed under Chapter 518, Laws of 1909 (N.Y.).

⁴Compulsory law Chapter 674, Laws of 1910, N.Y., and Voluntary Plan of Workmen's Compensation, Chapter 672, Laws of 1910, N.Y.

⁵*Ives v. So. Buffalo Ry. Co.*, 210 N.Y. 271.

Prevention

As to the first, prevention: it is far better for everyone, if the accident is prevented. We all talk of the preventable accident or the preventable occupational disease. Not all accidents or occupational diseases are preventable. But as to the preventable injury we need education, foresight, leadership, and constant vigilance. It was recognized early as one of the prime motives for the compensation system, even in the nursery we were taught that "an ounce of prevention is worth a pound of cure."

In England, before the compensation day had dawned in the United States, we find this principle espoused by the highest and most responsible statesmen. Speaking in the House of Lords on July 20, 1897, in a speech advocating adoption of the 1897 English Workmen's Compensation Law, Lord Salisbury said "To my mind the great attraction of this bill is that I believe it will turn out a great machinery for the saving of life. This is the real history of this law of compensation."

So completely had the principle of accident prevention been accepted that it was first written directly into the constitutional amendment adopted by the legislature and the people of the state of New York in 1913, authorizing the compensation laws. That amendment opened in this wise "Nothing contained in this constitution shall be construed to limit the power of the legislature to enact laws for the protection of the lives, health, or safety of employees, and for the payment," etc. of compensation. It enumerates the further powers necessary to provide for the payment of compensation for those injuries, regardless of fault, which despite preventive measures do occur, for security, and for everything else necessary to make the system workable.

Compensation is not designated as a punitive measure to be imposed upon employers who fail to prevent industrial injuries, although in effect it does that through in-

creasing the cost of compensation insurance. This suggests, so far as awards of compensation for cardiovascular diseases are concerned, that the cost to employers in many far-fetched cases only theoretically or abstractly related to the employment or any incident thereof takes on the aspect of a penalty imposed upon the employer for conditions beyond any possible preventive measure and conditions over which he has no control. This fear of the excess cost of the hiring of known cardiacs often operates as an almost insuperable obstacle to employment, and constitutes a hindrance to their rehabilitation.

Only when employers learned that under compensation laws accidents were costly to them was there any consistent or intelligent approach to the question of accident prevention. It would almost seem that, under the early concept, human life was somewhat lightly valued. Perhaps that aspect of the value of life and limb stemmed from the wars that recurrently, wave upon wave, swept over all Europe and, in fact, all over the world, in which a man's value was principally as a unit in the fighting forces of his country or of his feudal lord.

So long as the employer was required to make reparation only in case of his proved negligence, and then only when the three strong common law defenses were satisfied, the employer and his business could afford to take chances, and not until later years did he seek security in insurance for that limited liability. But when negligence and fault on the part both of the employer and the employee were eliminated, business found that the most effective way to keep costs down was by good safety practices. Out of that fact was born the great safety movement, and safety engineering became an honorable and necessary profession. Many industries have reduced their accident ratios and their efforts have been most rewarding. I venture that with all the high-speed machines, the utilization of vast power, and the great increase in the number of employed people, industry would grind to a halt because of the loss of life and serious maimings were it not for the measures planned by the safety engineers and effec-

* Hansard, *Parliamentary Debates*, 1897, Vol 51, p 555

ively carried out by management and labor co-operating together. Thus the compensation system, with all its preventive measures as well as its benefit provisions, has become an essential of the industrial system, and is constantly proving its worth by instilling safety into the minds of both management and the workers.

Curative Function of Workmen's Compensation

The second aspect of the compensation system is the curative function. I place it next in importance to the prevention of accidents and diseases because here, again, industry could not survive in its present state without the intelligent and skillful services of the physician and the surgeon and his whole-hearted co-operation. In no other respect is the difference between the old and the new systems pointed up so obviously as it is in relation to medical and surgical care. Under the common law and under employers' liability statutes, no obligation was placed on the employer to furnish any kind of medical care, although, under employers' liability insurance contracts, there was usually some provision to cover first aid in case of accident on the job. If a recovery was had against the employer, that recovery was based in part upon the cost to the injured person of medical and hospital care. Other than that there was no liability.

However, the most important aspect of the accident is to restore the injured person as promptly as possible to his former earning capacity or to the greatest earning capacity of which he may be capable in his physical condition. Of course no one in his right mind would trade off his earning capacity for the moiety he would receive in compensation. Nor would anyone trade off a member, i. e. a finger, hand, or an eye for the compensation provided by law for the loss of any such member. The loss of an anatomical member or the function of an ear or a vital function is beyond monetary calculation or price. Such loss can be judged in compensation only by the resulting impairment in physical efficiency measured by earning capacity.

Few doctors in practice today have had any experience in the treatment of a worker seriously injured on the job in the days before compensation. It meant time-consuming care and effort, most often on the promise of the injured man to pay if and when he could, with the right of the doctor to share in the recovery or settlement to the extent of his charges. Often, however, the case was settled behind his back. If he was called to come to testify, he had a good chance to receive something on account of his bill provided his patient got a verdict. But no verdict, no pay! This kind of practice quite understandably did not attract the most skilled men in the profession. The care the worker received was largely charity work, the hospital might collect a part of its bill, but the doctor, very seldom.

All that was changed by the introduction of the compensation system. When liability for accident was no longer dependent upon fault, medical care and treatment were also recognized as the obligation of industry. Medical service is not provided in the same manner or to the same degree in all jurisdictions. Limits in length of treatment or in an over-all maximum in dollars are common restraints. In a few jurisdictions, medical care is required without limit except that the charge shall be reasonable and the service necessary. In many jurisdictions the employer, who is charged with the cost of treatment, is given the legal right and responsibility of selection of the doctor, with appropriate provisions for those cases where he declines to furnish a doctor or where the treatment is not adequate. In other states we find the injured employee given either complete freedom or a limited right in the selection of the doctor to treat him. Where such complete freedom of selection exists, it has been found necessary to provide a standard authoritative fee schedule in order that the employer, who pays the bill, may not find himself helpless in a situation where the injured man, who does not pay the bill, might engage the services of a doctor of unreasonable character or questionable skill.

It appears that the majority of medical

bills in compensation cases come within the limits imposed by law where limits are expressed, and, to that extent, we may be confident that prompt and reasonable medical care is being rendered. At the same time we must recognize that serious cases do arise where the doctor is paid up to the limit provided in the law, but the injured person may still be urgently in need of further treatment. There should be, as there is in some states, at least the right of the doctor of the injured person to apply to the state authority for an extension of authorization for further treatment. We have seen innumerable cases where a stubborn infection or some physical condition requires treatment far beyond the time limited by the statute of the particular state. Streptococci do not read law books!

The accomplishment I am most proud of, during the time I was Industrial Commissioner of the State of New York, is that upon my recommendation, with the agreement of labor, management, and the Medical Society, the Legislature amended the law to place the obligation on the employer to furnish medical treatment and care for an unspecified and unlimited time. That was in the year 1922 and, since then, the New York law has required medical treatment for as long a time as the nature of the injury and the process of healing may require. For many years, the doctors and the hospitals have received full payment for their services and accommodations, provided the bills are rendered in accordance with the charges set out in the fee schedules or agreements and the cases are such as come under the law.

Doctors like the system because, totally unlike the old days under the Employers' Liability Law, they now get paid in every proper case. Medical expense is paid now in the State of New York to the extent of many millions of dollars each year. This in turn leads to better understanding and fuller cooperation between the doctors, the workers, the employers, and the insurance carriers.

Rehabilitation

The subject of rehabilitation must be regarded as a definite part of medical treat-

ment, although much of it is not of a strictly medical nature. In the sense that rehabilitation is the restoration of the injured person to the greatest usefulness in industry of which he may be capable, it is medical care and, as such, becomes the obligation of the employer or his agent, the insurance carrier. Rehabilitation calls for evaluation by the surgeon at as early a date after the injury as is possible. In fact, it should be in the mind of the surgeon from the start. Often it happens that a choice of operative procedure may be involved: which operation will offer the best opportunity for job resumption and greatest earning capacity? Some contend, and I think rightly so, that the job of rehabilitation restoration begins with the first treatment but that is not the general practice. Unfortunately, too many cases are allowed to run on for long periods without the start of definite rehabilitative procedures.

In an address in St. Louis in 1949, on the occasion of the Thirty-Fifth Anniversary of the International Association of Industrial Accident Boards and Commissions, at which I had the distinguished honor of being the guest speaker to review the accomplishments of the first thirty-five years of the compensation era, I referred to rehabilitation in the following language:

In the development of the compensation system, we have made astounding progress in many of its aspects. But in one field we have lagged and have somewhat "dragged our feet." That is the important field of rehabilitation. I have been hearing talk of rehabilitation for years and here and there something has been accomplished besides talk. But, by and large, we have accomplished little when measured by the needs and the opportunities. There has been talk of rehabilitation that misses the mark completely and is little or no better than good medical treatment.

Rehabilitation is not just medical treatment. It is training; it is the intelligent approach to each case as presenting its individual needs, it is, if you will, applied psychology. It calls for leadership, the inspiration of the injured to help himself, the instilling in the injured person of confidence in himself, and a feeling that after all he has a useful place in the world. It cannot be forced on anyone. You can't rehabilitate a man

who is determined not to be rehabilitated. Fortunately, there are not many who, when the system is rightly explained, do not want to improve their lot and strive faithfully and tirelessly to accomplish their physical and emotional training. But they need guidance, encouragement, and a type of leadership that has too frequently been lacking. Men need centers of rehabilitation—not homework. They do their best, when working somewhat in groups. Friendly contacts and friendly rivalry are important means to best results . . .

If private industry and insurance, yes, and self-insured employers, do not undertake an intelligent program of rehabilitation, the State will step into the picture, putting up the money, which it will raise by taxation of industry, and monopolize the whole field. *We cannot go on adding to the constantly growing human scrap pile without doing something effective to reclaim those who are capable of being reclaimed.**

In the years that have elapsed since these words were spoken, much has been done to advance the whole cause of rehabilitation. The subject of rehabilitation is a chapter unto itself and time does not permit of a full presentation in this paper. I can do no more than indicate the importance of the subject, and speak of it as one of the great factors in the problems presented by the workmen's compensation law. It is highlighted by the imperative needs of the millions of people who have cardiovascular diseases.

Of course, rehabilitation is not a problem of workmen's compensation alone. It is equally important for the cripple whose incapacity results from the automobile accident or the all-too-frequent home accidents or, again, from the devastating diseases involving paralyzes, spastic manifestations, or nerve injuries. These people, too, must be reclaimed and made useful citizens to the fullest possible extent. However, these problems do not impose statutory obligations on employers or compensation insurers and need not be dealt with here.

Rehabilitation of the Cardiac Worker

However, there is one aspect of the rehabilitation problem that should be mentioned here. It relates to the cardiac worker. The American Heart Association has a Committee on Rehabilitation, which functions as a unit of the Division of Community Service and Education of the Association. This committee has made studies and has encouraged the establishment and operation of Cardiac Work Classification Clinics. Skilled personnel in these clinics, including doctors, industrial experts, and employment advisors, make studies of the physical capabilities of recovered cardiacs, make work evaluations, determine the kinds and limits of work activity that may safely be undertaken, and aid in the placement of persons with heart disease. The work of the Rehabilitation Committee is integrated and coordinated with the work of the Committee on the Effect of Strain and Trauma on the Heart and Great Vessels, whose chairman is Dr. Paul D. White. This committee has undertaken a project for the study of the medical-legal, insurance, and industrial aspects of cardiovascular diseases. The Chairman, Dr. White, has long been an advocate of work for the cardiac within his capabilities. Dr. White said, a little more than a year ago: "A second consideration which I would like to emphasize is the beneficial effect of work on body, mind, and soul in any occupation in which it is possible for a cardiac patient to engage. Idleness breeds unhappiness and is actually bad for the health. It is a rare patient, indeed, who is fit for nothing. It greatly pays to make every effort to find something, either vocation or avocation, into which to fit the sick man or woman."²

This aspect of the problem of rehabilitation of the cardiac has received very wide discussion. It is a dramatic aspect of the problem. Here it is possible for the layman to be most helpful. It is not to be thought,

* Address, H. D. Sayer, *Proceedings of the IAABC*, St. Louis, Mo., Oct. 3-6, 1949. U.S. Dept. of Labor, Bureau of Labor Standards, Bull. 119, p. 33.

² Paul D. White, M.D., "Rehabilitation in Heart Disease." Presented at the Annual Meeting of the American Heart Association at New Orleans, October, 1955.

of course, that lay people, entirely on their own and without medical guidance, should attempt to give recovered cardiacs jobs for which they may not be fitted by reason of their physical condition. But employers should be encouraged to give work, if they have it, to recovered cardiacs, preferably with the advice and guidance of the medical advisor. Moreover, it is well for the attending physician to keep in the mind of his patient the thought of becoming well enough to resume work, so that he may lead a happy, contented life, notwithstanding his heart disease.

Not every job is good for every recovered heart case. Here is where the work classification unit can do its most useful job. Extension of the number of such units would seem to be urgently needed. They are available to employers willing to give employment to a cardiac, and can advise, after examination and work evaluation, the kind of job for which the applicant is fitted.

Compensation

What to many is regarded as the most important aspect of the compensation system is the compensatory side. It is the aspect commonly spoken of, and is looked upon by most people as the whole sum and substance of compensation. In the first place, it touches upon the lives and the happiness of the greatest number of persons, for every compensation claimant there are probably an average of at least three or four persons dependent, in some measure, upon the compensation paid. Secondly, it is the money part of the system: the money paid by employers directly or through insurance, and the money received by the injured persons for themselves and their families, or by the widows and children of the men who are killed. Thirdly, it is the tangible, demonstrable evidence that is ever in the hands and in the minds of the recipients. Week by week, as the instalments of compensation accrue, its importance in the way of keeping the family together and of providing all the necessities of living is most apparent. At the same time, as healing goes on and the

services of the doctor are needed less, the importance of the doctor's part in the situation is forgotten. Especially is this true if he is not the personal selection of the claimant. It is a curious phenomenon, but one that most administrators have observed, that often the injured person is a very different person at the time of his first treatment from the one he becomes later when he is no longer worried or frightened. At the time of injury his thoughts are centered on recovery. He tells the doctor in meticulous detail what happened and is frank and truthful in his past history. He wants to confide in the doctor and have confidence in him. After he has recovered from the first shock of injury and has time to think about his future, he becomes a claimant, often with a completely changed point of view. His recollection of events sometimes becomes dim and confused. At the time of the settlement of his case it is to be expected that he and his representative, if any, will try to maximize his injury. The employer, on the other hand, who at the time of the accident may have been most solicitous and insistent that the best of care be provided, may later take a different attitude when the final settlement is under consideration.

Every safeguard should be employed to prevent the accident. However, when despite all care and precaution an accident occurs, we look to the physician to care for the injury, to see that the care is adequate and promptly furnished. After medical care has been rendered, or at least started, we look to the compensatory side. Here we find the greatest variation in the laws, in procedures, benefit levels for payments, and in many other details. Notwithstanding differences, we find much that is alike in the compensation laws of all the states.

We have seen from the foregoing something of the history and background of workmen's compensation, what is supplanted, and what it was intended to accomplish. In its relief provision it was designed to afford a speedy, informal, nontechnical procedure in lieu of the former controversial, litigious, and highly technical system.

The dominant feature of compensation is the absence of fault as a cause of injury. This does not mean that every injury, regardless of cause and its relationship to employment, is included as a compensable injury. There are proper and necessary limitations as to the injury that may bring it within the purview of the law. These limitations are expressed in the well-nigh universal provision that injuries are covered if they "arise out of and in the course of the employment." This simple clause is gravid with meaning. It would seem at a glance to be simplicity itself, yet it has been the provision of the compensation laws most prolific in the production of litigation and court interpretation. This exact formula or its practical equivalent is found in all compensation laws. A few states have omitted from the formula the requirement "arising out of", it is considered sufficient that the injury "arise in the course of" employment. The difficulty has been to determine precisely what acts are committed and what circumstances exist only because of the employment and just what is intended by "in the course of employment." Actually, almost all injuries to workers do occur in the course of employment, that is, during the hours on duty and at a place where their work requires them to be or ordinarily takes them. Questions may arise as to whether an employee has stepped out of his employment for the time being, abandoned his work to go to some place to satisfy his natural curiosity, for enjoyment, or for something in the nature of a personal errand. A simple example might be an employee sent out on a job who took time off on his way back to watch a ball game, and was struck on the head by a batted ball. Travelling between home and work raises difficult questions at times. While generally "going to work" accidents are not deemed in the course of employment, there may be circumstances where they might be covered. An example is the salesman who may be in his employment continuously while he is on the road, practically a 24-hour exposure.

The many cases where controversy exists as to whether a happening arises out of the

employment are more complicated. The law books are replete with court decisions on some of the most bizarre and fantastic events that have occurred in the employment but as to which there may be a very real question whether or not they were work-caused, including all manner of events that are normal and incidental to the daily life and contacts among workers. There are wide variations in the attitude and interpretations of commissions and courts on some of these questions.

It seems to me that these words "arising out of and in the course of employment" have other significance too. From time to time, arguments are heard for liberalizing the compensation laws, increasing benefits to unrealistic figures seemingly without regard to the ability of employers individually and industry as a whole to meet the increased costs imposed thereby, widening the scope of the laws and their coverage, and, in effect (whether so intended or not), transforming the compensation system into a general health and accident law for workers. That is not the philosophy of compensation, at least as far as its original concept and background are concerned, nor the basis upon which it was accepted by the people. There are some who advocate limiting coverage for injuries only to those arising "in the course of" employment. The relation of the injury to the employment need not be apparent, it being sufficient to locate the accident or the onset of the disease to time and place that brings it chronologically and physically within the employment. The formula in most of the existing laws is "arising out of and in the course of employment." These two clauses are conjunctive and to satisfy the law both conditions must be met. The plea for this new concept seems to be based upon the postulate that workmen's compensation is a social instrumentality, having little regard for the matter of the employer's liability. I think the facts set forth above amply demonstrate the fallacy of that concept.

I would not assume to discuss here whether there should be such a social insurance. If it is decreed by the people

that they want such a law, it should be enacted directly, and not made a charge upon a system that was otherwise conceived and adopted by the people. The maintenance of such a system of social relief becomes a matter of taxation. The compensation premium dollar is not a tax. It is the contribution by each employer toward the relief of injuries for which industry is directly liable, and is proportioned to the number and cost of injuries sustained by his employees and employees in the same type of operations. This contribution is a charge upon production and, as such, is met by each and every one of the users of the product or service. Therefore, when the total of all costs of production rises to a point where the product or service is priced out of the market, what becomes of our employment? When employment cannot be maintained because of high production costs, the worker out of a job becomes our first concern and he is the first to suffer.

It may not be amiss to mention, at this point, that it is a truly American principle to accept the compensation laws in each state as being considered by the people in that state as being best suited to the particular needs and customs of their industries and people. There have always been those who advocate the centralization of all authority in the central government, in the compensation field we have not been without such wishful thinking by some. We should view the compensation laws of each state as representing the majority thinking of the people in that state—labor, management and the people generally, who in the last instance pay the cost of all compensation. No two states are exactly the same in their climate, their natural resources, their industrial and agricultural potentials and opportunities. It is, therefore, not surprising that we should have many diversities in our state compensation laws. Uniformity of the laws is not the primary consideration, and uniform provision in all states could lead to some difficulties not now envisioned.

Other fundamental principles and philosophy of compensation will be found in

the fact that benefits are not only payable promptly as they accrue, but that people shall be treated alike in the same circumstances. To effect such treatment, it was provided wisely that benefits shall be paid in accordance with certain formulas. The equation reflects the earnings or the earning capacity of the individual, ascertained in each case in accordance with the applicable law. Benefits are payable for the period of disability, whether the disability is total or partial, temporary or permanent. In the fatal case benefits are payable in accordance with the relationship of survivors and for such periods as each state may provide. Unlike the old system of jury trials, benefits are not left to the judgment of jurors or, more frequently, to the guess, whim, or caprice of the jury. Once the entitlement of benefits has been determined, the rate will be mathematically determined in accordance with the law. The period for which benefits are payable becomes a medical question within the limits of the law and will be determined by medical evidence of disability.

Disability

What constitutes disability? Do we always distinguish in our minds whether we think of disability in the economic sense or in the anatomical or physiological sense? Various of the laws declare that compensation shall be paid during the continuance of disability, usually with an over-all maximum limit or with a maximum expressed in dollars. In general, disability is accepted as a condition resulting from an injury that incapacitates the worker from earning wages such as he earned at the time of or prior to the injury. It may be total, that is, inability to work and earn any wages, or it may be partial, that is, inability to do regular work and earn full wages but with ability to earn some wages at a less laborious job. In such case, the rate of compensation benefits is proportioned to the actual earnings or reduced earning capacity. Wages and capacity to earn wages are the two essential factors in the measurement of benefits to be paid.

Difficult questions arise from time to time

and confusion is sometimes introduced by reason of considering disability on a purely anatomical or functional basis, without regarding wage-earning capacity as the true test. For what do we compensate—an anatomical and physiological impairment that may not result in any loss of earnings or earning capacity? Or do we mean disability in the sense of economic and wage loss? In the various laws, disability and the right to compensation have been related to wage-earning and earning capacity. This is the so-called "wage loss" concept. Of late years, it has become the fashion to urge that the wage-loss principle is all wrong, that something must be paid, and paid generously, for cosmetic defects, for functional disturbances such as partial deafness, with no loss of wages or working ability, and any condition causing mental distress, on a purely physiologic or anatomic basis. More and more, age and degenerative changes are becoming directly or covertly asserted as compensable disabilities under the law. The so-called "schedule loss" is cited as the authority for bringing such conditions under the law regardless of wage loss. This argument does not satisfy me. It seems to assume that the schedule provides a determinable payment for an anatomical loss regardless of its effect on work ability. Such is not the case.

We use the term "schedule loss" familiarly and frequently, but what does it signify? A schedule loss is a bodily loss that assumes an actual existing or potential future loss of earning capacity, the compensation for which is measured by a schedule set forth in the law. A loss that is scheduled is the loss or loss of use, either complete or partial, of a member of the body, the loss or loss of use of which is permanent but not resulting in total disability. Such members are ordinarily specified in the law, the loss of an eye, arm, hand, leg, or foot or part thereof. The loss is permanent in the sense that it cannot be fully repaired or replaced. Compensation is properly payable for such a loss. But how may such a loss be measured in terms of disability?

It is of interest to note that in the early

English law, upon which our laws were largely patterned, there was no schedule loss. The injured worker was paid for actual disability only, and the disability paid for was that immediately following the injury during which the worker had to stop work. When the worker was able to return to work, his compensation stopped. One way considered by those who drafted the law was to pay compensation for life (since the loss continues throughout life) but at a lesser than the full rate, such rate being determined as such proportion of the full rate as the actual disability might bear to total disability. This idea was not generally adopted. Instead the practice adopted and written into the law in most states was to pay the compensation at the full rate but for a proportionately shorter period of time. The schedule is designed to reflect the relative industrial importance of the member lost. For instance, the loss of an arm, concededly the most serious loss of any member so far as working is concerned, entitles the injured person to compensation for 312 weeks in many states. These schedule allowances are proportionally scaled down until we find that the loss of a little finger calls for compensation for only 15 weeks. This compensation is payable when the defect is finally determined without regard to whether the injured person presently loses time from work or not, although, in any serious case, it is a practical certainty that he will lose time from work until the site of the injury has completely healed and he returns to work.

Why is this done? It will not be denied that the amputation or loss of use of a member will more or less seriously impair the working capacity of the worker. For that he should, of course, be compensated. He may be able to do everything in his work that is required or, as often happens, the employer keeps him on, after the healing is complete, and makes due allowance for the reduction in his productive capacity. However, his job may not always be available to him and, in the course of time, he may have to seek work elsewhere. His handicap then is real and his disablement becomes very important in

a work sense. The time of this job loss may be only weeks later or it may be years after the original injury. His case cannot be held as an open case indefinitely for payment of actual disability compensation. The employer may no longer be in business or work may be slack, his insurance carrier could not be expected to put aside a large reserve for each such case and carry it on the books perhaps for years, as it might have to do, if the case were not officially closed.

So it was determined, and wisely so, that the disability measured by the reduction in his earning capacity is conclusively presumed, and that disability is compensated in advance of an actual demonstration of a loss of earning, now for then, so to speak. However, the compensation rate is always a function of wages at the time of injury, and the compensable period is always expressed in terms of weeks at the wage rate.

It is a completely different legal concept from the theory of *damages* for a wrong done. The discretionary lump sum compensation for facial disfigurement is pointed to as an illustration of the nonwage-loss compensable disability. However, even in the case of the compensation for disfigurement we must not overlook the fact that the justification for a facial disfigurement award is that it may be so unsightly or repulsive that it may have a marked effect on wage-earning capacity or getting a job. It is not just a generous handout. The allowance for serious facial or head disfigurement was no part of the original compensation system and was a subsequent departure from the original purpose and intent of compensation recognized only in some states. It surely is no justification for a radical departure from the intent and purpose of the compensation system as it was represented to the people.

The rates for compensation insurance are a function of wages in the various types or classifications of hazardous employment and reflect the cost of injuries in each classification. Wages and the wage-loss concept are the only reasonable, the only fair, and the only practical measure of compensation liability.

Disability and Compensation in Cardiac Cases

This discussion of schedules and wage loss may seem far removed from any consideration of the heart in industry. But is it? How are heart cases that occur among workers and related to work, actually or seemingly, compensated under the workmen's compensation laws? There is a great variance in practice in the states; some are very liberal in the interpretation of the law and in the establishment of accidental disability whereas some others are more technical in the requirement of proof. Whatever may be said of the fatal case of heart failure and the adjudication of cases involving such claims, a very different situation is presented with respect to disability in the nonfatal case when the claimant recovers from the acute condition presented by his heart attack. Is his condition permanent and is it permanently and totally disabling? Can a legal conclusion on this question be based upon such proofs? Clearly, even though the condition be adjudged to be permanent, it cannot be made a basis for a schedule award. Should it be legally regarded as a schedule and awarded as a schedule injury? This could not possibly be done. In a number of states, settlements on a lump sum basis are made in heart cases and death cases are often settled at a fraction of their full value because neither the claimant, the widow or children, nor the employer is sufficiently sure of his position to be willing to try out the issue in a legal proceeding. Hence the settlement. Personally, I do not like the idea of such settlements; either the claim is good, in which case it should be paid in full, or if the heart condition was in no way attributable to the work, no payment should be made. There is no middle ground.

If the heart case is likened to a schedule injury, then the payment, if any, should close the case. In the death case, the payment, made on an agreed basis, would completely release the employer. However, in the disability case, where payments are made over a period of time, a very difficult question may arise, if the disability ends in death. Then the question arises anew, whether the

terminal result can be attributed to the original work incident that made the disability case compensable. These are but illustrations of some of the imponderable questions that are precipitated by consideration of the heart case under workmen's compensation laws.

Security for Compensation and Administration

We have touched upon some of the fundamentals of compensation as it is found in the United States and the philosophy underlying them. Two points remain concerning which some comment may be useful. These are security for compensation and administration of the laws. Compensation is not paid or payable in a lump sum, as a rule, with a discharge of all further liability upon such payment, as in a case at law. On the contrary, the accepted basis for compensation payments is periodically during the continuance of disability. In theory, and in fact, the extent of compensation payments cannot be determined until after all medical care has been rendered and the worker is able to return to work. That would mean, in many cases, waiting a long time to adjudicate and adjust claims. So periodic payments start early and continue regularly during disability. The liability of the employer for these payments extends for years in many cases. The solvency and business stability of the employer are hazards that the injured employee, or the widow and young children of the deceased employee, cannot rightly assume. Therefore, the obligation of the employer must be supplemented by the obligation of an insurer, be it a state fund, a private insurance company, or a qualified self-insurer. Insurance and self-insurance perform a necessary and vital function in the operation of any workmen's compensation law. Through such means the liability of the employer is supplemented and his liabilities taken over completely by his insurance carrier. It is the service and the solvency of insurance that makes possible the operation of the law and the assurance that any seriously disabled person or dependent of any deceased person will receive the periodic payments of the compensation to which he or

they are entitled, no matter for how many years the claim may run. There are many cases today in which payments are being made and have been made for more than 25 years. The reserve funds to continue such payments are required by the law to be maintained at all times. Thus security for the employer's obligation is made certain.

The magnitude of the problem of workmen's compensation is apparent in the number of workers covered by the law in one state or another. The Federal Security Administrator in 1953, after a most careful and painstaking study, estimated the number of workers in this country who are subject to workmen's compensation to be 39,000,000.¹⁰ In 1956, the Secretary of Labor of the United States issued a public statement in which his Department calculated that the number of persons gainfully employed at that time was 66,500,000. The number of persons presently covered by workmen's compensation is somewhere between these two figures.

Another feature of workmen's compensation is the matter of the administration of the law. The general rule, with but few exceptions among the states in this country, is administration and adjudication by boards or commissioners who form a part of what we have long recognized as *administrative law*. Four decades or more ago, our common law or statutory courts were being swamped with actions at law brought against employers by their employees. Court proceedings were long delayed, and many cases of hardship occurred where no real money was paid out for two, three, or more years after the accident. A principle of compensation was developed whereby prompt payment was made possible, not of a large final sum, but periodic payments of compensation made much as wages, promptly and as they accrued. Trials by jury were eliminated. Simple and nontechnical procedures were established. Administrative officials were clothed with power to hear and determine the facts of claims and to make legal conclusions. In getting at the facts, technicalities and tech-

¹⁰ *Social Security Bulletin*, 17 2-12, March, 1954.

nical rules of evidence were pretty generally done away with. Hearings were held before these officials where necessary. Different forms of procedure were authorized in different states. Generally, the findings of fact of the administrative officer or board are final and may not be reviewed by the courts on appeal. Appeals to the courts from such officials are generally permitted, but only on questions of law.

In some few states adjudication of claims and enforcement of orders is charged upon the courts. Upon a petition or an action at law, such cases are brought before the court in a prompt and summary manner. Characteristically, we may say that administrative law prevails in the vast number of cases to the extent that it may be regarded as the characteristic procedure. Differences are found as to many administrative matters. Variations are found in methods of payment, in hearings and adjudicative procedures, as to lump sum settlements, as to appeals, and, very importantly, in the matter of statistics and statistical reportings.

DISCUSSION

Looking back on my more than 40 years of close association with workmen's compensation, I am proud of the development of this great compensatory, preventive, and curative system, and of the opportunity I have had of participating in it. It has worked mightily in the public interest. While accidents may have increased in number, it is certain they have been held down in the face of the greatest upsurge of industrial activity and industrial production in the history of mankind. The national cost to industry has been in the billions of dollars over the past 40 years. It has extended relief to untold millions of persons and has made possible the development of industry. Three years ago, the benefits paid in compensation to claimants, exclusive of medical services, were calculated under the New York Workmen's Compensation law, from its inception in 1914, down to December 31, 1952 at the sum of \$1,594,702,376 by insurance carriers

alone.¹¹ A possible 10 per cent would have to be added to these figures to include payments by self-insured employers.

To these figures must be added millions of dollars to cover the costs in other states. I would be a daring man who would attempt to calculate or estimate the total benefits paid out in the nation as a whole, including private insurance, state funds, and self-insurers, in addition to the government, both state and federal. It staggers the imagination to think of the billions of dollars of relief afforded.

To these monetary benefits should be added the cost of medical service to the millions of workers who have received, without cost to themselves, the finest medical and surgical treatment accorded to workers in any part of the world. Medical and hospital payments at the cost of industry may conservatively be estimated at billions of dollars, in addition to other benefits in the more than 40 years of compensation.

This is the system of the compensation liability of United States industry. This is the system that, as a service to industry, has kept the wheels of industry turning at full speed with the greatest possible protection to the workers. We have not, however, satisfactorily solved the problems that arise in the heart case. These problems fall mainly into two classes, the fatal case in which the problem is principally concerned with the determination of liability, and the nonfatal case, which presents not only the problem of liability, but also the problem of disability and the even more difficult problem of rehabilitation and employment.

As to both classes of cases, we should strive for some general understanding of the conditions antecedent to the fatal or crippling attack, which may or may not have come about during work. It is important to have some clear thinking on the problem that is often presented as to the influence of work on a diseased heart or blood vessels, whether the work constituted the usual and ordinary activities on the job or whether there

¹¹ 40th Annual Report of New York Compensation Insurance Rating Board, 1954, p. 7.

some unusual event. Need the unusual event be something unexpected, unforeseen, accidental and fortuitous, or may it be something of a common expected occurrence? We cannot put our whole reliance upon court decisions in these problems, for the courts are confined largely in their review of cases to questions of law, not questions of fact. If there is evidence in the record that supports the conclusion reached by the administrative authority, no matter how fanciful or far-fetched, the courts as a rule will not, perhaps cannot, disturb the administrative findings. Sometimes we find court decisions that seem to be utterly conflicting, when the difference may have been in the purely fact-finding function and decision of a board or commissioner.

In the nonfatal case, where the worker has made a satisfactory recovery from the immediate effects of a heart attack, we are met with a very different situation. The heart case in compensation is a high-cost case. That cost has an immediate effect upon the rates that the employer must pay for his insurance, if he is insured, and it has a very direct and continuing effect on the self-in-

sured employer. The employer who has experienced an increase in his insurance costs by reason of heart cases understandably, perhaps unfortunately, reacts to protect himself by refusing employment to workers with a known history of cardiovascular disease. This conflicts with all our hopes of rehabilitation of the heart case. If employers are to be severely penalized in their compensation costs by reason of being held liable for disease conditions that have been acquired over the years and over which the employer or the employment had little if any control, some method would seem to be necessary whereby the cost may be spread over the industry, rather than be charged wholly to the immediate employer. This, however, raises a subject to be discussed by others at this conference rather than in this paper.

It will not be denied that the heart cases under compensation have a profound effect upon our industrial life. The extent to which they have developed in recent years compels us to give heed to the problem and to dedicate our best efforts to seeking a solution, fair alike to industry, to the workers, and to the whole public.

CHAPTER 51 CAN WE REHABILITATE WORKMEN'S COMPENSATION?

Kenneth E. Pohlmann

When the opportunity was afforded me to participate in this Panel on Workmen's Compensation of the First Wisconsin Conference on Work and the Heart, I accepted reluctantly. Knowing how well Mr. Jerome Pollack could have arisen to this occasion among such an impressive list of participants, my feelings of inadequacy increased in geometric proportions as the deadline for the delivery of my remarks came nearer. However, taking a lesson from my initial experience in learning to swim, I said to myself, "You're in it up to your neck, and you'd better thrash about if you want to stay above water." You can judge whether or not I've sunk after reviewing my remarks.

Dr. Belknap, in his letter of invitation, made it even more difficult for a reluctant participant when he emphasized that "this conference will not deal with generalizations. It will bring together qualified people interested in the entire field of work and the

heart, and offer an opportunity for frank discussion and interchange of ideas." The words that stuck in my crop were, "qualified people," because by no stretch of the imagination or propping up with pillows could I fit into that category. The opportunity for "frank discussion and interchange of ideas" was another thing again. Here, I thought, is the one place where a nonexpert could get in a word edgewise if he waited for an opening, and was quietly persuasive.

To begin with, my inability to pass on the merits of medical arguments in the field of cardiology as they relate to workmen's compensation is even more limited than that of a compensation hearing referee who, on the basis of conflicting medical testimony, *can at least, and often does*, rule in favor of the claimant. I do recognize, and I am sure others do also, that among physicians of unquestioned competence there often exist differences of opinions. It was not too long

ago that it was commonly held that exertion directly contributes to coronary disease—now there is a swing of opinion which holds that it doesn't. But this is not likely to be the last oscillation on this and other medical matters relating to compensation. As recently as March, 1956, there appeared an article¹ which in a small way helps to support the often-expressed claim, even among physicians, that the practice of medicine has not yet become an exact science. This article also minimizes the role of heart claims in the over-all claims picture under workmen's compensation in that state. Perhaps we need further to examine other state compensation programs in the same manner, and learn whether or not we are making a mountain out of a molehill. Too often we tend to deal with issues in nongquantitative terms. From such labors we may develop opinions and even policies, but the net result is that we are not, in this manner, meeting basic problems.

In the workmen's compensation field we operate on the assumption that injuries and diseases can readily be sorted out into non-occupational and occupational classifications. This is a fine distinction that no longer basks in the aura of infallibility, thanks to the fact that claimants' attorneys are practicing medicine and some physicians, in a pinch, deem to practice law. I believe, on the basis of reading many hundreds of cases in the course of my work, that there now exists a less firm medical base for workmen's compensation decisions than has been assumed.

We are dealing, in workmen's compensation, with an ailing form of social insurance, conceived in an era when "the capacity of the economy to support an adequate remedy for work injuries was presumably not yet demonstrated, when social legislation was in its infancy, and when the status of the industrial worker left much to be desired. The laws born under these circumstances had acknowledged congenital defects, the early benefits were very modest; serious restrictions were placed on allowances for long-term disability and death; serious restrictions suffered from an

which they have never recovered;* and the usual plans of administration were not equal to the task posed by the legislation."² The history of workmen's compensation during the past four decades is replete with earnest attempts by compensation administrators, forward-looking labor leaders, competent medical authorities, and enlightened management, to bring it into focus with the changing industrial and social order, but it has remained, for the most part, bogged down in a morass of piddling reforms, always too little and too late. It has yet to catch up, in many states, with the diseases contracted in new industrial processes, to award physicians and hospitals their just dues for services rendered, or to reimburse the industrially injured worker anywhere near his lost weekly wages, or to pay his survivors anything but a very modest death benefit.

The sad plight of this first social insurance scheme on the American scene has long been recognized by those who administer it in the various states—the members of the International Association of Industrial Accident Boards and Commissions—who, year after year, in their annual conventions soul-search for answers to their problems. They cannot be accused of not having some of the answers, for, as Dr. Herman Somers told them at their forty-second Annual Convention in Charleston, South Carolina, last December, "the records of your proceedings are enough to disarm any critic. Whatever shortcomings the outsider may point to in workmen's compensation, you can show on the record that you said it first!"

In many respects workmen's compensation has turned out to be a bad bargain for labor in return for giving up the right to

* I
the
19

mon, declared
law of
uting
t occu-
rity

sue the employer for negligence in the courts. Undoubtedly, when the program was originally proposed the worker was with few exceptions in an especially disadvantageous position, with low wages, a minority status in society, and little if any political power. However, it is hard to believe that the proponents of workmen's compensation could be as shortsighted as they have turned out to be in failing to bring this program in line with an expanding economy, the improved status of the worker, and the knowledge we have (for instance) of rehabilitation techniques. It is becoming increasingly evident to many injured workers that, with a weak workmen's compensation program, they might be actually better off to resort to common-law suits since, in general, workmen's compensation serves more to *limit the employers' liability than to protect the worker*. The creation of the National Association of Claimants' Compensation Attorneys, probably inevitable under the circumstances prevailing in many state compensation jurisdictions, and the success with which its forays into the courts on cases have met, leads one to conclude that the old farmer was right when he said, "Problems have an uncomfortable habit, if they are not solved, of solving them who is responsible for creating 'em."

Workmen's Compensation's failure to keep pace with progress does not foredoom it to oblivion, but it is later than we think to legislate the basic reforms necessary to bring it into tune with the times. Too many years ago, I remember sitting in a hearing room in a state legislature listening to testimony for and against certain proposed changes in workmen's compensation statutes. It is interesting to note how little the scene has changed in the decades that have intervened; the same forces for change, the same crowd against, despite the tremendous advances we have made in medical knowledge, rehabilitation concepts, and the like. Certain reforms have finally been proposed by two outstanding medical groups, the American College of Surgeons and the American Medical Association, which could go a long way in meet-

ing the prevailing deficits in workmen's compensation. I commend them to your serious study since, with your professional standing in the community and the nation, an earnest effort on your part to co-operate with other elements in the population interested in improving workmen's compensation, would do much toward breaking down the opposition to change altogether too prevalent in this field.

Many of the current victims of inadequate workmen's compensation benefits become the public assistance cases of the future. We either meet the problem when we are faced with it or it comes to haunt us at a later date. There is a rich field of study open to some bright researcher as to what happens to serious accident and disease cases subsequent to adjudication of their compensation status. Certainly, the story, from my observations, has been a sad one for the severely disabled coal miner.

The interest of all segments of the population of the United States in social insurance, whether voluntary or compulsory, has grown at a phenomenal rate since the 1930's. The depression years had a profound effect upon the American people that no amount of Madison Avenue "hoopla" to the contrary can now completely eradicate, since the population group of the depression years is now the older-age group of today and the decades ahead. They have experienced social security, unemployment compensation, Blue Cross, Blue Shield, and other forms of insurance against disease and dependency; they have been impressed by the productive capacities of our United States industrial system and our farms; many of them know that our nation is committed through the legislative and executive branches of government to a full employment policy. They may talk glibly about too much government, but in a showdown they urge their legislators to support measures for improving social security, expanding health facilities and services, and for doing something for the older-age population. The phenomenal growth of support for the rehabilitation activities of the Federal-State vocational rehabilitation pro-

gram and the fact that it was the one item in the Health, Education, and Welfare Appropriation Bill substantially unaltered by an economy-minded Congress should give all of us cause for reflection. Perhaps the American people, through the orderly democratic processes, are "cutting themselves in" on some of the benefits that our human and natural resources have developed.

Organized labor, through collective bargaining, has negotiated income benefits and health insurance for nonoccupational diseases, at a much faster rate than they have made gains in workmen's compensation. It is not inconceivable that, in time, this expanding field may simply take over most, if not all, of the workmen's compensation liability. In fact, an eminent cardiologist at the meeting of the International Association of Industrial Accident Boards and Commissions at Charleston, South Carolina, in 1956 even went a step further in suggesting that in the cardiac case it might be desirable to develop a disabilities benefit program, ostensibly through government, to relieve the workmen's compensation system of the burden of such cases—a more radical departure, please note, from that of any spokesman for organized labor! In theory, it would appear that organized labor originally supported the concept of separate accountability for work injuries on the part of the employer in order to have the economic incentives for safety and occupational hygiene stemming from such an arrangement. In practice, merit rating leaves much to be desired. The costs of workmen's compensation, on the whole, are really negligible when compared with other fringe benefits and social security. (Currently, employers are paying a little more than half of 1 per cent of payroll for workmen's compensation.) This low level of expenditure, with relatively unimportant variations—and I might add in passing that the heart disease component is entirely negligible as a cost item—has reduced the effectiveness and significance of merit rating. This may steam up a few of my fellow panelists but I throw it in for what they want to make of it.

In the Middle Ages, I am told, theological debates reached the stage where eminent clergymen worked themselves up into a lather over the question as to how many angels could dance on the head of a pin. In some respects, we, in the most highly industrialized nation in the world, are emulating these medieval purists, we are doing our damndest to establish a dichotomy of diseases and injuries, one group occupational, the other nonoccupational, one compensable, the other not. In the process we have lost sight of the basic problem. What happens to the man? To his family? And, at what cost to our society?

The effects of dependency and disease upon our economy occupy volumes of testimony before congressional committees, our educational institutions are filled with learned tomes documenting the close relationship between dependency and disease, and our colleges graduate hundreds of people each year who are supposed to be qualified to cope with these problems. A whole new language, sprinkled with cleverly phrased clichés, has emerged in psychiatry, social work, and related disciplines, to describe the ills that flesh is heir to, but the injured industrial worker, the superannuated employee and their families still must have the means to purchase groceries, to pay rent, and to clothe their nakedness. Whether or not these essentials are provided through such benefits, unemployment compensation, disability awards, grants-in-aid, or just plain poor relief is not for the purposes of argument essential, but it is essential that they be provided.

It behooves us then, it seems to me, to examine workmen's compensation as part of a total group of services available to the disabled worker. He may need additional services, not provided through existing state compensation programs, if he is to overcome his disability and, perhaps, return to productive employment again. Some of these services, including prompt payment of weekly benefits, physical rehabilitation, guidance and counseling, retraining, and job placement are now available to injured workers through certain insurance carriers who pro-

vide "extra coverage" for added premiums. They can be provided through co-operative arrangements between state workmen's compensation agencies and the state vocational rehabilitation programs. Some self-insurers have demonstrated the value of providing to injured or diseased workers prompt treatment and after-care, under competent medical management, beyond the legal requirements of the state workmen's compensation statutes, with resulting re-employment and a salutary effect upon the morale of the entire work force. Labor-management agreements exist in many areas that provide for special consideration being given to re-employed disabled workers with regard to seniority rights, job classifications, and the like. It goes without saying that such negotiated contracts presume that adequate first aid and medical and related benefits will be provided the worker from the time of injury until return to work.

Claims adjusters, solo practitioners, and others dealing with industrially injured or diseased could learn something from the experiences of such carriers as the Liberty Mutual Insurance Company, the American Mutual Liability Insurance Company, and a handful of others, who have pioneered either through their own resources or through such facilities as the Kessler Institute of Rehabilitation, the Institute for the Crippled and Disabled, and the Institute for Physical Medicine and Rehabilitation, in bringing to bear upon the problems of the injured industrial worker the latest that medicine and the related disciplines have to offer. Organized medicine, in its zeal to maintain free choice of physician, except for the injured industrial workers under workmen's compensation, has not always looked kindly upon group practice clinics and teamwork concepts, as these techniques have come to be used in the practice of medicine and in the furtherance of rehabilitation. Some insurance carriers, through their medical departments, have had real difficulty in persuading practicing physicians to accept additional medical services or procedures in compensation cases where it appeared that prolonged

treatment was neither benefiting the patient nor the carrier. In short, the strength and support given to rehabilitation, both medical and nonmedical, has come in large measure from outside the medical profession, although with the enthusiastic co-operation of a small segment of outstanding medical leaders.

The impact of rehabilitation upon workmen's compensation practices is just beginning; the "parochialism" of state workmen's compensation programs still presents some real barriers to the more effective use of rehabilitation techniques in getting a job done, but there are many forces at work among insurance agencies, medical groups, and particularly within labor and management circles which are tending toward more hopeful possibilities. Concurrently, the infiltration of rehabilitation concepts into voluntary and official social welfare programs, some of the newer public health activities, the cumulative effects of the Federal-State vocational rehabilitation program, and the President's National Employment of the Physically Handicapped program, are giving real momentum to the attack upon disablement and dependency.

The time has long since passed when piecemeal solutions are applicable to the problems of disablement and dependency. The problem of the patient with heart disease, as with any other disabled person, compensable or not, is no longer *only his concern* or an isolated problem with a "pat solution." Whether or not he makes the readjustment necessary once again to establish himself as a productive worker in society has become the concern of many forces, and a vast new group of services and facilities are becoming available to assist him in so doing. Our society can no longer exist half dependent and half productive. Inheritance taxes have taken care, in part, of one group in the population who are nonproducers by accident of birth, and newer health and welfare approaches are being developed to rehabilitate those who are nonproducers because of injuries or disease.

Out of the productive capacities of her people, the United States has been shaping.

slowly but surely, constructive measures for meeting the needs of those who have been struck down by disease or disability. For those who cannot take their place in the production line, the hopelessly crippled and disabled and the older worker, we have disability insurance and old-age and survivors' insurance. Some segments of our population will continue to resist such efforts, but so long as the United States pursues the high purposes implicit in the democratic process, we can believe that we shall con-

tinue to find hope and new opportunities for our diseased and disabled

BIBLIOGRAPHY

1. BEARD, R. R., BRESLOW, L., THOMAS, W. H., GARDIFEE, C. R., BUECHLEY, R. W., and MULLINS, V. W. Heart disease claims under the California Workmen's Compensation Act, *Circulation* 13 448, 1956.
2. POLLACK, J. A policy decision for Workmen's Compensation, *Industrial & Labor Relations Review*, Vol 7, No. 1, October, 1953.

CHAPTER 52 WORKMEN'S COMPENSATION AND ITS RELATIONSHIP TO THE HEART

Theodore C. Waters

You will pardon me if I begin my presentation in the first person because I would like to identify myself, my background, and my relationship to the administration of compensation laws. You will find that I am prejudiced in some of the views hereinafter stated.

As a lawyer, I have always represented insurers and self-insurers under our workmen's compensation laws, except in those cases where employers have employed me to represent their employees in claims against the company. At the trial table I have the employers' point of view. Therefore, you may justifiably consider that my views are prejudiced in their behalf. In preparation for this meeting, I had read countless opinions of Commissions and Courts dealing with the subject of our discussion. It is not my purpose to deal with those decisions in detail because that would only mean confusion confounded. American industry faces a serious problem in dealing with compensation

cases involving the heart. We will not find all the answers to this problem during the course of the excellent program that has been prepared for this meeting. However, I believe that the meeting affords an opportunity to bring out into the open some of the basic issues involved in the matter of workmen's compensation in heart cases.

My professional practice has been limited to a few states from the eastern seaboard, Maryland, Virginia, West Virginia, New Jersey, Pennsylvania, and New York. There are others far abler to present the legal details of issues in these cases presented to our Compensation Commissions. However, I do have a few thoughts that may represent a contribution to our consideration of this complex problem.

In addition to my study of Commission and Court decisions involving heart cases, I have also read a number of articles dealing with the medical, legal, and industrial aspects of our subject. Again, it would be im-

possible within the time allotted to me to attempt to make more than brief references to these articles. Only to mention the names of Dr. Paul D. White, Dr. Elston L. Belknap, Dr. Leonard J. Goldwater, Mr. Benjamin E. Kuehle, Dr. Rodney Beard, Dr. Charles D. Marple, Dr. John W. Ferree, Dr. Leo Price, Dr. Henry Ungerleider, Dr. Norman Plummer, Dr. Howard B. Sprague, and Messrs Barnett S. Fox and Henry D. Sayer, makes brief reference to those who have contributed so much to our thinking in this matter. I cannot refrain from reference to the excellent papers presented at the recent meeting of the International Association of Industrial Accident Boards and Commissions in Charleston, South Carolina, last December by Doctors T. W. Mattingly¹ and Richard J. Clark.² All of us should read and reread these valuable contributions. The American Heart Association and those doctors who are specialists in this field have done much to clarify our thinking. The vice of the matter in my thinking is that the legal profession, State Commissioners and Courts, while willing to receive that advice, do not properly evaluate it, with the result that those of us in my profession are permitting workmen's compensation statutes to become health insurance statutes and imposing upon industry the legal obligation to provide compensation for all ills to which the human flesh may be subject, irrespective of the relationship between the happening of an accident, the potential aggravation of a disease that existed prior to the happening of the accident or whether or not the *injury* that is the subject of complaint arose out of and in the course of employment. Unless and until our Commissions and Courts are properly educated with respect to this matter, the existing chaos in the administration of compensation laws will continue.

If it is socially desirable, let us amend our laws, divorcing our legal thinking from the concept of workmen's compensation, and impose upon an employer the obligation of awarding compensation for any and all illnesses, irrespective of the causative factor of that illness and irrespective of the issue as to whether or not it arose out of and in

the course of employment. Query: Do we propose to make our workmen's compensation laws health insurance laws? If so, let us call them that and not delude the employer and the public by misnomer of the statutes that may be involved.

Turning now to the second person, may I direct your attention to certain basic fundamental principles involved in workmen's compensation. These statutes were originally designed to remove from our common-law courts disputes arising between employers and employees for industrial injuries. The original concept contemplated coverage for accidents, that is, trauma occurring at a specific time and at a specific place and while the employee was in the employ of a specific employer. By judicial construction and legislative amendments these statutes were extended to cover occupational diseases. What do we mean by the term "occupational disease"? While this term is subject to many definitions, the one that appeals to me is a disease that is characteristic of and peculiar to a given employment. It is needless for us to be reminded again that by Commission decision and Court construction many diseases of human life that *may* be contracted by the employee's extra employment have been the subject of awards of compensation under our statutes. Is this theory to be extended to heart cases? What criteria will the medical profession adopt as a guide to our Commissioners and Courts as causative factors or aggravation of existing heart impairment? What criteria will the medical profession adopt as the basis for recommending that certain heart cases should not be compensable under our statutes? What will our Commissions and Courts do with respect to the acceptance of these criteria? What will become of the mechanics for the disposition of heart cases by our Commissions and Courts in ruling upon the compensability or noncompensability of heart cases? To state these questions of necessity indicates the magnitude of the problem facing us. Again to return to fundamentals, may I call your attention to the definitions of the terms "injury" and "disability" as construed by our

Courts There is no uniformity in these definitions. Counsel may readily find numerous definitions that may suit the particular claim involved in the case at bar. Do not think that I am critical of my brothers on the other side of the table representing claimants. It is their duty to represent their respective clients to the best of their ability in an effort to attain the objective for which the claim is filed. My criticism is directed to the status of the law, the attitude of Commissioners, and the attitude of our Courts. In the administration of our compensation laws throughout the nation we face a growing tendency to consider the compensation laws as the media for doing everything that can be done to assist the claimant in receiving compensation for the injury complained of. Personally, I am in sympathy with this general philosophy but I am critical, should I say supercritical, of our Commissions and Courts that place upon the employer and insurer the heavy burden of disproving beyond all reasonable doubt the compensability of a given claim.

At random, I have chosen the following definitions of the term "injury".

"Injury" as used in Workmen's Compensation Act and as applied to a human being, includes whatever change in any part of the system which produces harm or pain, or lessens the facility of natural use of any bodily activity or capability. *McLean's Case*, 93 NE 2d 233, 234, 326 Mass. 72.

Again, we have the following definition:

In common speech the word "injury" as applied to a personal injury to a human being, includes whatever lesion or change in any part of the system produces harm or pain or a lessened facility of the natural use of any bodily activity or capability. *Furlong v O'Hearne*, DC Md, 144 F Supp 266,270

Acceleration, aggravation, or lighting up of pre-existing disease as a result of employment is "injury" for which full compensation is recoverable for entire disability suffered. *Tanenbaum v Industrial Accident Commission* 52 P 2d 215, 216, 152 Or 205

Here is a typical case with the construction of the definition of the term "injury":

An employee, helping to erect a stone crusher, made several trips in carrying water in buckets, and then undertook to carry, from a wagon to a car, bags of coal each weighing approximately 150 to 200 pounds. The first to be taken was handed to him and carried to the car. The next bag was rested by the passer on the rim of the wagon wheel. The employee reached to take it from the wheel, and a minute later he was lying on the ground in a dying condition. The medical examiner testified that the employee's heart muscle was tired and exhausted at the time of his last work, and that his final exertions caused the inability of the heart to perform its work. Testimony of two physicians justified this assumption. Held to sustain a finding that the death of the employee was caused by "injury", within the Workmen's Compensation Act. *In re Fisher*, 108 NE 361, 220 Mass 581.

Summarizing my own thinking as to the legal aspect of this term, I would say that our courts generally construe the term "injury" to mean "harm to any part of the body."

Let us now turn to the concept of the term "disability" as used in our compensation statutes. Here again we have countless definitions for that term and I will call your attention to only a few. Before doing so, however, I would like to revert to the originally basic purpose of the compensation statutes relating to compensation for "disability." As we all know, the amount of compensation proposed to be payable has always been stated in the terms of a percentage of the loss of wage. This is peculiar to all our statutes and follows the precedent of the laws adopted by the English House of Commons, which form the precedent of the adoption of our laws.* Therefore, it is only fair to state that we started out in this country with the purpose to relate compensation for disability arising out of and in the course of employment to the wages earned by the employee during the course of his employment.

* See Dodd's *Administration of Workmen's Compensation*, p 16

Again, we find that our Commissions, Legislatures, and Courts have diverted from the concept. In summary, three distinct concepts of the term "disability" are used by our Commissions and Courts:

- 1 Inability to earn full wages.
- 2 Total inability to perform any other work
- 3 The event of an employee becoming actually incapacitated from performing his work in the last occupation in which he was engaged

Again, we face a tendency on the part of administrative agencies and courts to divert from the principle of wage loss and to compensate for "injury" irrespective of wage loss. To be brief, I have faced this result in my own professional practice. In those states where I have practiced and perhaps in many others, industrial commissions have become identified with a most unpleasant name, "gift shop." In the administration of compensation laws, some states permit trial *de novo* before juries and we find ourselves governed with a new phase of common-law administration, except that the employer is stripped of all legal defenses. The sole issues are the following.

- 1 Whether or not the claimant sustained injury arising out of and in the course of employment
- 2 The nature and extent of his disability

Does it make sense for cases of this type to be tried before juries with attendant prejudice to the employer defendant? My answer to that question is "No." To revert to the term "disability," I quote the following random concepts of various courts

The word "disability," as used in Workmen's Compensation Law, means impairment of earning capacity, and not loss of a member, and is that which disqualifies an employee from doing work in whole or in part. Comp St 1929, sections 48-101 et seq, as amended *Wilson v Brown-McDonald Co*, Neb, 278 NW 254, 261, 116 A.L.R. 702

"Disability" may result as well from the condition of the mind and nerves as from other causes, and where a man is so inattentive or forgetful as a result of mental disorder that he cannot be trusted to carry on even simple forms of work he is as "disabled from earning a livelihood" as one who must refrain from work on account of the condition of his vital organs. *United States v Taylor*, CCANC., 110 F.2d 132, 134

"Disability" within Compensation Act occurs when employee is disabled from rendering further service by present physical inability to perform work in usual and customary way, and in absence of such disability, employee sustains no compensable injury though employment may have subjected him to exposure which contributed to ultimate disability from occupational disease. St 1931, §§ 102.01, 102.03(1) (a) *North End Foundry Co v Industrial Commission*, 258 N.W. 439, 217 Wis 363

The test of "disability" under the Louisiana Workmen's Compensation Act is whether employee can do same type of work he was doing at time of his accident in the customary way without any unusual difficulty or pain. *Strickland v. W. Horace Williams Co*, CALa, 230 F.2d 793, 797.

The fact is that we face a growing tendency in the administration of compensation laws to treat claims for compensation as claims for damages and, in my opinion, this is grossly unfair to employer, employee, and the general public.

Unless and until this trend is reversed, the potential of claims under our compensation statutes is staggering. Take, for example, claims involving injury to the heart, aggravation of the heart, or the sequelae of heart disease.

Indicative of the significance of my last statement, I can only refer to the excellent paper prepared by Dr. Mattingly,⁴ in which he stated and I now quote

Finally, there is one type of heart disease included in the so-called unusual causes of heart disease which is very important to you—namely that of traumatic heart disease. In the present era of high-speed transportation and mechanization of industry, there is an increase in the types of

injury which results in direct trauma to the heart and great vessels. Where there are direct injuries of the penetrating type to these structures, such traumatic lesions are readily appreciated and appropriately handled. With nonpenetrating injuries and indirect injuries, however, the etiological relationship to trauma is often overlooked, either at the time of injury or when latent complications occur from such injuries.

In a recent study of 207,548 autopsies on file at the Armed Forces Institute of Pathology, evidence of trauma to the cardiovascular system was found in 1,125 autopsies or 0.5 per cent, and was considered severe enough to cause death in only 0.1 per cent of the total studied.

There are no existing data known to me as to the proper percentage of heart cases that should be compensated, but I know and you know that compensation is being awarded in many of these cases where there is no direct causative relationship between employment and the occurrence of the heart attack.

In my remarks so far, I have attempted to deal with certain basic philosophies with respect to compensation, knowing full well that many in the audience, or those who will read the record of this proceeding, may be in disagreement with some of the views that I have expressed. However, I have no apologies for the statements made nor do I mean to minimize the importance of this problem to employers, insurers, and employees. Having stated the problem, I have asked myself and this panel, What are we going to do about it?

The Utah plan with which we are all familiar has been carefully considered by various groups. While I am in sympathy with that plan, I am uncertain of its legality. It is not within my province to express an opinion as to the criteria established by the Utah Heart Association and the Salt Lake Society for Internal Medicine for compensation in heart cases. My good friend, Otto A. Wiesley, the Commissioner in Utah, has done a stupendous job in his effort to resolve this problem. Properly an autopsy is required in all fatal cases. I am an advocate of the plan to provide medical experts to advise the

Commissioner in controversial cases, but this presents legal problems that may well defeat the operation of the plan and would certainly meet with great resistance in other industrial states. However, something can and must be done about this matter. Perhaps legislative changes, medical panels may be granted the necessary powers properly to adjudicate the medical issues involved in heart claims. As an advocate of such panels and medical boards, I have met with repeated resistance to this subject in various state legislatures in the east, not only by representatives of employees and employers, but also representatives of insurers and self-insurers. That criticism, however, has only strengthened my belief in the social need for the resolution of medical questions by a competent, totally independent Board of Medical Examiners completely divorced from any interest in the case at bar. I have such high regard for my brothers of the medical profession as to believe that they can and will effectively handle medical issues involved in any given claim. Basically, they are far more competent to serve this function than a lay commissioner, lawyer, judge, and certainly a jury. If I am ill and consult my doctor, who diagnoses my case and prescribes treatment, I do not then ask lay persons, or twelve men tried and true, to tell me whether or not my doctor was correct in his opinion. It is admitted that some panels or boards and some doctors will make errors. We are all human, but should strive to attain the best possible administration of our compensation laws in the disposition of controverted medical issues. The fact is that in the trial of most cases involving medical issues, some doctor may be willing to testify for a given claimant or for an employer and express such opinion as his patient, or counsel for his patient, may desire. Assuming that there is medical testimony in a given case to support a claim, the tendency of an administrative agency is to award compensation for some amount, even though the most reputable physicians in the given locality would testify that in their opinions the injury did not arise in the course of and out of employment. Our courts

follow the general principle that if there is evidence in the record to support a claim they will not disturb the decision of the Commission, even though the personal opinions of the jurists may differ. Again referring to the Utah plan, I would like to make brief reference to a rule adopted by the Court of Appeals of Maryland granting authority for the appointment for neutral medical witnesses by trial judges in controverted cases. For the benefit of the record I quote the following Discovery Rule 5-1 of the General Rules of Practice and Procedure now effective in my State, applicable to all courts of the State:

Whenever the mental or physical condition of a party is material to any matter involved in any proceeding, the court may, upon motion by any party and notice to all other parties, for good cause shown, order such party to submit to a mental or physical examination by a physician or physicians. The order (1) shall specify the time, place, manner, conditions and scope of the examination and the person or persons by whom it is to be made and (2) may regulate the filing of a report of findings and conclusions and the testimony at the trial by the examining physician or physicians, the payment of the expenses of the examination and any other relevant matters.

Commenting on this rule, I quote the following comment in the *Daily Record*, a Baltimore newspaper, under date of Tuesday, January 11, 1955.

Although the court may not have power in the absence of an application by one of the parties to appoint an expert witness, in most cases where there is wide disagreement between the parties as to the extent of a medical disability, it will probably be found that one of the parties is anxious to have a neutral expert of high standing appointed, and is willing both to make application for such appointment, and to guarantee the payment of the expert's fees. Whether or not to grant the application is in the discretion of the trial judge. The power of the judge to select an expert of his own choice and without the agreement of counsel as to his identity is confirmed by a number of decisions in jurisdictions in which a similar rule is in force.

We are blessed in Maryland with two of the nation's outstanding medical schools: Johns Hopkins University and the University of Maryland. It has been my privilege to have served for some years on a Medical Legal Committee of the Maryland and Baltimore City Bar Associations, and the Medical & Chirurgical Faculty of Maryland, our State medical society. You may well ask as to the practice followed pursuant to the adoption of this rule. The Chief Judge of the Supreme Bench of Baltimore City, Judge Emory H. Niles, has expressed himself publicly advocating the use of the rule. However, as a matter of practice, we find that in the absence of agreement among the parties to given litigation the courts will not appoint independent medical experts. Unfortunately, in the greater majority of cases, counsel primarily, or litigants secondarily, will not agree to the appointment of such experts. Therefore, we are left with the administration of the law wherein Dr. A for the claimant-plaintiff expresses one opinion, presumably liberal to the plaintiff, and Dr. B for the defendant-employer expresses another view entirely at variance with that of Dr. A and presumably liberal to the defendant. How can a court or jury properly evaluate such testimony?

Having posed the question, I shall attempt to answer it in a few words. In my opinion, the only method to correct this situation is by statutory enactment. Having appeared before various legislative committees as an advocate of this plan, and having had my ears pinned back time and time again, I am dubious of successful effort in this direction unless and until the general public becomes educated to its importance and insists that their duly elected legislative representatives should take affirmative action. Perhaps the most significant role to be filled in this connection is that played by our medical societies. They have or should have the confidence and the respect of the various communities in which we live and upon them rests the primary burden of taking the initiative to the end that some solution to this problem should be found.

Perhaps I have gone somewhat afield in the subject that was assigned to me, but I would like to close with one further thought

Again, I pose a question What about the employee who has an existing heart condition? Is he disqualified from employment? Are employers willing to accept him knowing that they will pay a potential compensation claim? Must he be relegated to the human scrap heap and denied the right to work? Why do not our brothers of the labor movement recognize the significance of this problem and join with employers, medical groups, and the legal profession to find a reasonable, constructive answer?

It has indeed been a privilege to participate in this panel discussion. I know that I have profited by information received from other participants whose views I respect,

even though I may disagree with them as well as having them disagree with me.

The Committee sponsoring this Conference deserves the thanks of the general public and through the media of such conferences we may look forward to the day when the problems of cardiacs in industry may be resolved

BIBLIOGRAPHY

1. MATTINGLY, T. W Panel: Medical aspects in determining responsibility in heart cases, pathogenesis of heart disease (as related to employees compensation), *Proc Internatl. Assn Indust. Accident Bds and Commissions*, U S Dept. Labor, Bull. 192, p 103, 1956
2. CLARK, R. J The heart and workmen's compensation, *Proc Internatl. Assn. Indust. Accident Bds and Commissions*, U S Dept Labor, Bull 192, p 120, 1956.

CHAPTER 53 MEDICAL WITNESSES IN WORKMEN'S COMPENSATION

Ivan C. Rutledge

The essential characteristic of workmen's compensation schemes has been their elimination or de-emphasis of the notion of fault on the part of the employer.¹ One of the intended effects of the elimination of questions of fault was to accelerate the disposition of contested cases, as well as to make it easier for the workman to obtain benefits in at least some amount. Questions of fault having been either eliminated or largely reduced, the focus has shifted to medical questions: the nature of the injury or disease, its relationship to the employment, and its disabling effects. In the current outlook of our secular

society, there is no authoritative morality except as it is pronounced in law by politically selected officers, so questions of fault are peculiarly adapted to resolution by adversary litigation. In medicine the contrary is true, the authority of Science tends to sustain the pronouncements of one who can claim its authority on the strength of his profession. Processes of litigation as we know them today were developed in the context of questions of fault, but such questions have receded in workmen's compensation and although litigation was transferred to newly invented tribunals, it was preserved. So it is that litigation has been preserved for the resolution of medical questions, for which it is ill adapted. It is not necessary to demonstrate that litigation looks most clumsy when it undertakes to deal with questions of expert opinion.² It may be possible, however, to

¹ An alternative emphasis has been placed upon their function as part of a system of income protection by insurance, a philosophy especially germane to calculating the monetary level of benefits and calculating disability by occupational rather than physical or mental impairment. See Arthur Larson, "Changing Concepts in Workmen's Compensation," 14 *NACCA L Jour* 23 (1954); Stefan A. Riesenfeld, "Contemporary Trends in Compensation for Industrial Accidents Here and Abroad," 42 *Calif L Rev* 531 (1954).

² For example in *Flores v State*, 236 Ind 151, 168, 139 N.E.2d 185, (1956) at 196, the court approved this instruction: "the law of Indiana recognizes temporary insanity. You are

examine the administration of workmen's compensation to determine what, if any, opportunities it may afford for somehow avoiding the notorious battle of the experts.

Three parts of the problem may for convenience be taken as ways of seeing into it. First, the determination of the nature of an injury or disease with particular reference to its connection with an employment, and to some extent with reference to the extent of its disabling effects, depends in large part for its reliability upon the accumulation and preservation of information upon which medical inferences leading to the determination must be based. Second, the reliability of the determination may, in some of the most difficult cases, equally depend upon the quality of expert opinion brought to bear on the data that have been accumulated and preserved. Finally, even with the most thoroughgoing processes of investigation and recording, and the employment of the best current medical opinion, the final results may be unsatisfactory unless the method of organizing the processes by which expert opinion is brought to bear upon the data are such as to provide safeguards against its misapplication. These aspects, then (1) the accumulation and preservation of information, (2) the mobilization of expert opinion, and (3) the organization of the methods by which it is brought to bear upon the information, are the three windows chosen here for a view of medical testimony in the administration of a plan of workmen's compensation.

Although questions of fault are less prominent and questions of medicine more so under workmen's compensation plans, it would be a mistake to suppose that the con-

clusion should be in favor of placing their administration in the hands of medical referees or panels. Perhaps the most significant argument for lay administration can be suggested by relating an episode in *Jenkins v. Industrial Commission*.³ A workman was handling an automobile battery when it exploded, causing him to jerk his head back suddenly and slap at his eyes with such force that the frames of his spectacles were bent. Three weeks later an operation to correct a separation of the retina in his right eye failed, and he subsequently received compensation for loss of vision in the right eye. Four months later a physician observed loss of vision in the left eye, which he estimated as 75 per cent (in terms of loss). In proceedings for compensation for injury to the left eye, the commission appointed three eye specialists to examine the record in the case and give an opinion on the relationship of the accident to detachment of the retina in the left eye. In their report they said:

The files state that Mr. Jenkins (1) was a high myope, (2) had bilateral cataract extractions, (3) had bilateral capsulotomies, and (4) had peripheral retinal degeneration of the left eye between 5 and 7 o'clock with many vitreous floaters, as indicated in Dr. Irvine's report . . . 4 months after the injury, and at which time no evidence of detachment was found.

We, the undersigned, feel that (1) the above findings are more contributing causes of a detachment than was the indirect injury, and (2) since no evidence of detachment was found 4 months after the injury that the resultant detachment 2 months later, or 6 months after the injury, was due to pathological disorganization of the retina rather than to trauma. We feel that the Commission was very generous and liberal in accepting the detachment of the right eye as

not to be influenced by the statement of any medical physician who testifies, if they testify that there is no such thing as temporary insanity. There is a distinction between the definition of temporary insanity in the law and in medicine and it is the law in Indiana that if a person is temporarily insane at the time of the commission of the alleged act then you must find him not guilty." (Of course no court would so reprobate medical testimony

that there is no such thing as being bewitched, even if the code denounced witchcraft as a crime. Cf. *Reg v. Machekequonabe*, 28 Ont. Rep. 309 (1898)—taking judicial notice that there is no such thing as a Wendigo, or evil spirit clothed in human form which eats human beings.) See Thayer "Trial by Jury of Things Supernatural," *Legal Essays* 325 (1908).

³ 77 Anz. 377, 272 P.2d 601 (1954).

compensable as all these types of cases present an element of doubt. We are of the opinion that the detachment of the left eye is not related to his injury whatsoever. (Emphasis added)

On the other hand, the other doctors (who testified by deposition) found a probable connection between detachment of the left eye and the injury. No point is made here whether obtaining an opinion by written report is less reliable than obtaining an opinion by testimony subject to cross-examination. It is also unnecessary to characterize the language italicized above as representative of medical opinion in any degree. The question is how this language aided the triers of fact to reach a decision. On the one hand it suggests that the commission had erred in the other case and that both cases were subject to the same or a similar degree of doubt. Thus the expression can be taken as a way of advising the commission about the degree of doubt that obtains on such an issue as a general scientific fact. On the other hand it suggests that the commission should make compromise awards in doubtful cases. That is, when there is doubt whether several injuries should be attributed to an industrial accident, compensation should be awarded for less than all of those injuries instead of resolving the doubt as to each of them. This interpretation of the italicized language amounts to an interpretation of the statute, which is not within the special competence of a medical expert. It therefore casts doubt upon the reliability of the medical opinion given, that the separation of the left eye was not connected with the accident, because this may be a legal, or medicolegal, rather than medical conclusion.⁴

The use of medical experts as triers of fact is, of course, subject to numerous objections in addition to the risk that the medical opinion may be consciously or unconsciously affected or displaced by a legal opinion,

without adequate procedural machinery to determine the accuracy of the legal opinion.⁵ The day-to-day work of administrators of compensation, although it does not deal extensively with questions of fault, is devoted to administrative processes that much of the time require no medical expertise. Among these processes are those that serve the first function mentioned above, the accumulation and preservation of information. In the *Jenkins* case⁶ the history prior to the explosion was nearsightedness as long as the patient could remember, and an extra-capsular cataract extraction performed on the right eye five years before the explosion, with post-operative experience of irritation, redness, and pain for some time. The certainty of opinion could perhaps have been improved, especially with reference to the right eye, had it so chanced that medical examination had intervened between the operation and the explosion, and the nearer to the explosion the greater the probability of consistent medical opinion expressing a high degree of confidence in the existence or nonexistence of connection with the explosion.⁷

⁴ What professional qualifications could be hired for the salaries paid compensation commissioners? Would the possessor of medical qualifications be content to spend his days deciding issues that require no specialized knowledge and giving a fair hearing to the parties to those issues?

⁵ 77 Ariz. 377, 272 P.2d 601 (1954).

⁶ The function of expert opinion on the relationship between two events is to establish its existence or nonexistence within a described range of certainty. The trier of the fact is then to determine what has been established with what degree of certainty and resolve any doubts accordingly by a finding of fact or perhaps by a denial of a finding of fact. Legislatures and courts seek to maintain legal control over this process by the creation and application of presumptions and burdens of proof that prescribe degrees of certainty, such as "preponderance of evidence," "substantial evidence," "scintilla of evidence," "speculation and surmise,"

the process of resolving doubts that arise in the face of incomplete knowledge. This "resolution" of doubts after all available information is in, if deliberate rather than impulsive, can be thought of as a process of reasoning them away, or putting

⁷ The court, from a comparison of the opinions, directed the commission to set aside its order denying compensation, one justice dissenting. *Ibid*

ACCUMULATION AND PRESERVATION OF INFORMATION

Economic considerations limit the activity of collecting information that may improve prospects of certainty in determining causes of disability. It would be wasteful to conduct medical surveillance of all employees on a level that assumed that all of them would become parties to contested cases concerning their disabilities. A scheme of workmen's compensation does, however, apply to a situation that is clearly enough defined to make it administratively feasible to maintain a system of accumulating information. The existence of an employment relation itself yields certain records, such as payroll records, which may be sufficient to show days, and even hours, on the job. Administration of claims provides records for the future, of facts gathered on previous investigations. Public offices outside the compensation system, such as courts, public health and vital statistics and coroners' offices, may have information. Hospitals and medical and legal practitioners' offices are additional repositories, although some of this information may be confidential. Initiative of the employer in requiring medical examinations upon being hired and occasionally or periodically thereafter can give information that may become useful.⁸ This regime, because of

considerations of economics and employee relations, should be selective in approach, so that intensity and frequency of examination are guided by the apparent needs of the case. (For example, there may be a correlation between absenteeism and compensation neurosis.)

Many compensation systems include a requirement that the workman report the occurrence of an injury to his employer. Although by the time of the injury badly needed information may be lacking, it may have been impossible or impracticable to have exercised sufficiently selective prevision to have assured an adequate record of such information. Yet certainly upon the occurrence of an injury a specific signal for selection is important. It may be impossible then to gauge how important it is to select the injured employee for development of his medical history, but without the signal another opportunity for obtaining greater reliability of medical opinion will have been lost.

The main problem in administering this requirement is how much notice of injury must the employee have had before he is obliged to report it to his employer. In most jurisdictions the period within which the report must be made does not begin to run until the injury manifests disabling effects.⁹

them out of attention for purposes of decision or of concentrating upon them to the exclusion of inferences that might be drawn in their absence. The purpose for which the decision is to be made, e.g., payment or denial of payment, radical or conservative treatment or no treatment at all, further exploration to obtain further knowledge, etc., will influence the trier of fact, and as explained in connection with the *Jenkins* case, may influence the witness, regardless of verbal legal controls. Litigation is no less than a procedure for raising and evaluating doubts, and when witnesses anticipate the trier of fact by an opinion that evaluates what ought to be the disposition of the case as distinct from an opinion that describes the degree of doubt on the issue, the procedure is perverted. (The "ultimate fact" rule, now properly under general attack, presents a related question. See Ladd, "Expert Testimony," 5 *Vanderbilt L. Rev.* 414, 423-435 (1952), for a discussion leading to the conclusion that a violation of this rule is an "imaginary error".)

⁸ There is a reference to the "current hiring practice" of conducting a thorough medical examination and investigation of the medical history for latent physical conditions, in Comment: Injury to the Deceased or Disabled Employee under the California Workmen's Compensation Laws, 44 *Calif. L. Rev.* 548 (1956). The Comment points out that the rule in California gives impetus to this practice because it imposes risk of greater liability upon employers in cases where the workman is apparently well but is really not than in cases where he is obviously suffering some disability or where he is actually in good health and sound condition.

⁹ *Contra*, under the Longshoremen's and Harbor Workers' Act, at least when the workman is aware of some injury. *Pillsbury v. United Eng. Co.*, 342 U.S. 197 (1952). Other cases to the contrary are found in North Carolina, Illinois, Kentucky, and Pennsylvania, under statutes that prescribe the beginning of the period as the "accident." Oklahoma and North Dakota also start the running of

This means a possible time lag within which the opportunity to obtain valuable information is lost, unless there is an examination by chance. Moreover, the statutory period itself constitutes a similar risk of lost opportunities. The purposes of the requirement, however, to enable the employer to provide treatment that will minimize the seriousness of the injury, as well as to make possible an early investigation, must be balanced against the hardship¹⁰ that would be imposed upon the workman if a shorter period were given or if he were required to have a degree of foreknowledge of disability that could not be expected of him. The latitude afforded the workman by the requirement as it is usually made argues for a program of health and safety education on the job that includes encouragement of voluntary reporting to an office or aid station, where the information will be intelligently handled and followed up when professional investigation or treatment seems to be indicated.

The accumulation and preservation of information upon which expert opinion can be based begins in an intensive fashion when treatment begins, or at the latest, when a dispute about liability or extent or permanence of disability has reached such a point in litigation as to make it necessary. The most prominent issue for medical testimony is the connection between the work and the injury, with its disabling effects. Professor Small has emphasized that the consideration of employment as a "risk factor" in disablement is uncongenial to the medical discipline, which looks for "cause" in etiology.¹¹ The factors that range beyond pathology are outside the responsibility of the doctor as a scientist, although it may be that diagnostics can never entirely dispense with a degree of autonomous, free-hand detective work. It is

beyond the qualifications of the writer as well as the scope of this paper to explore the approach of either the clinician or the expert to whom a subject is referred for examination pending litigation. Two aspects of the problem of accumulating information at this stage must, however, be emphasized. One is the desirability of a high degree of completeness of investigation and the other is the necessity for systematic and durable records of the information obtained.

Ideally, when for the first time a potential medical witness examines a workman in the context of possible industrial injury or disease, existing information relevant to establishing what it is and whether it is connected with employment should be collected at one place. This clearing-house function is essential for purposes of litigation, even when diagnosis is obvious from objective findings alone, although in such cases (assuming that such cases occur) the history elicited from the workman may be summary and other types of information, such as that contained in hospital records or records of other doctors, may be explored only to the extent of identifying their sources. At the other extreme are cases in which the most sweeping and random techniques of exploring the past are called into play, the workman is searchingly cross-examined, the recollections and records of members of the family, associates on the job and elsewhere, other doctors and professional men, public offices, hospitals, etc. are searched, information is compiled in minute detail with rigid adherence to the form in which it was originally cast, the physical examination is detailed and comprehensive, employing tests and tactics that may be expensive and even impose risks on the workman, extensive resort is had to the laboratory, and specialists and consultants are

the period with the "injury" regardless of apparent effects. See 2 *Larson's Workmen's Compensation Law* § 78.42(b), pp. 264-265 (1952). The period ranges from 24 hours to 6 months when specified in terms of a quantity of time. A few jurisdictions further specify a maximum term to which delay may be excused, ranging from 30 days to a year. Some statutes say "immediately," forth-

with," or "as soon as practicable." *Id.*, App C, pp. 554-555.

¹⁰ "Shocking injustice," according to *Panchak v Simmons Co.*, 15 N.J. 13, 103 A.2d 881, 891 (1954).

¹¹ Small, "Gaffing at a Thing Called Cause: Medico-Legal Conflicts in the Concept of Causation," 31 *Texas L. Rev.* 630 (1953).

drawn into the investigation. Medical investigators will appreciate the significance of these two extremes because they superficially indicate the range of measures that are appropriate as guides for treatment. The prospect of litigation merely opens up more lines of relevance than when only the question of treatment is in view. The universe of investigation in workmen's compensation adds the question of employment as a risk factor in whatever disability may be feared from the injury or disease that is revealed.

Little needs to be added for present purposes concerning records of these investigations. The principle is that the memory of the examiner is unreliable (at least without means of *refreshing* it) and the labor that went into the investigation should not be wasted for lack of a system for finding the records, lack of a record of what could easily be recorded at the time, or lack of durability of the record.¹²

The object of gathering such information as will obviate unnecessary resort to inference, expert opinion, and surmise justifies the suggestion that medical practitioners will respond to a social obligation to find out whether litigation, either of a tort or workmen's compensation claim, is likely to be involved, and if so, to investigate for litigation as well as treatment. Of course it is the duty of the lawyer also to investigate for litigation, but his investigation will be more fruitful and litigation may be avoided more frequently if it can build upon prior medical investigation that was timely and under expert direction. The responsibility of the doctor to whom a workman is referred for examination in view of litigation is clear; and especially when the referral is partisan it is with equal clarity the duty of the lawyer to contribute what he can to extending the scope of investigation in the directions indicated by the needs of litigation.

The statutes generally require the workman to submit to examination, although they vary as between examination by a medical

practitioner selected by the board and one selected by the employer or insurer. Many of them are deficient in that they lack provisions requiring post-mortem examination and allowing for exhumation in proper cases.¹³ The importance of autopsy information is great enough to justify explicit statutory rules regulating the balance between the need for information and the sensibilities of the survivors.

MOBILIZATION OF EXPERT OPINION

"Our inexpert use of experts"¹⁴ presents the paradox of the layman evaluating the qualifications of the professional. The necessity for skilled assistance in the trial of a factual issue was born of an awareness that the common knowledge and experience of jurors would not be adequate to deal with the issue. This awareness and this claim of qualification under present practice arise from partisan assertion of the deficiency of common knowledge, and partisan proffer of qualifications that will fill the gap.

The issue of the need for expert assistance may be illustrated by *Thoreau v Industrial Accident Comm'n*.¹⁵ The workman in the course of his employment sustained a violent blow in the pit of his stomach from the end of a large timber. Ever after that time he suffered intense pain in the stomach area and was unable to keep food on his stomach or to do any work. A complete clinical review revealed to the specialist to whom he was referred a pyloric stenosis, or narrowing of the opening from the stomach into the small intestine. Now if a nonmedical witness had undertaken to testify to the existence of the pyloric stenosis, it would certainly have been required that he make some showing of his ability to perceive or infer it. This showing would have had to take the form of

¹² See *Petition of Sheffield Farms Co.*, 22 N.J. 548, 126 A.2d 886 (1956).

¹³ Weihofen, "An Alternative to the Battle of the Experts: Hospital Examination of Criminal Defendants Before Trial," 2 *Law and Contemp. Problems* 419 (1935).

¹⁴ 120 Cal. App. 67, 7 P.2d 767 (1st Dist. 1932).

¹⁵ See *Medical Economics* 66-67 for a 13-point checklist for records, quoted in *Physician in the Courtroom* 78-80 (Schroeder ed. 1954).

evidence of special experience, because it can be judicially noticed that common experience does not enable witnesses to detect pyloric stenosis. That is, the tribunal can judicially know the meaning of the term in a general way, sufficient to raise serious doubts, at least, whether a witness without special tutoring or experience would perceptually recognize a pyloric stenosis. It would probably be beyond all doubt to the tribunal that such a witness would be unable to find one by inferential means such as judging signs and symptoms or conducting tests. The existence of a broken finger might or might not be recognizable from common experience, and in this case the tribunal might require expert assistance to determine how doubtful the case was.

Thus the paradox of lay determination of the qualifications of experts is compounded by the paradox of lay determination of the need for experts. Although both issues are addressed by partisan assertion and denial, it can be seen that logically there is an infinite regression here: the determination of the qualification of experts or the need for an expert calls for expert assistance, which in turn calls for an expert to determine the expertness of the assistant, etc. Dean Wigmore characterizes rules of expert qualification and their administration as finicking matters that should be left exclusively to the discretion of the trial judge without appellate review.²⁸ And it seems to be generally true that this is the case, with the result that the qualification of an expert is rather a perfunctory matter procedurally unless the advocate who produces him prudently chooses to make careful adduction of his qualifications. The scene of combat shifts to efforts to disqualify particular answers and weaken the weight of the expert testimony given. Furthermore, in most cases, as in the case of the unobviously broken finger or the pyloric stenosis, lay knowledge is so clearly imperfect as to make the need for expert assistance apparent.

The pit-of-the-stomach case also involved an expert conclusion that the cause was un-

related to the blow and was attributable to a pre-existing ulcerous condition. So far, if there had been no blow and the pre-existing ulcerous condition had been patent, the lay mind probably would have reacted favorably to the suggestion that a doctor should be called in to verify the existence of a connection. However, it was taken as a fact that the ulcers, although they pre-existed the blow, were asymptomatic. (How this fact was established is not clear from the report of the case, and the reader is left to speculate whether timely investigation might have revealed a pre-existing pyloric stenosis.) The layman's *post hoc, ergo propter hoc* thus is bolstered by the evidence of a blow falling on the very part of the body that becomes symptomatic. Not much belief in witches is required to convert sequence into consequence here.¹⁷

There were two weaknesses of the expert opinion, one that the specialist made a written report and did not appear for cross-examination, and the other that the specialist was retained by the employer. The technique of the court was to find that the opinion was not based on a consideration of the fact that the existing ulcerous condition was asymptomatic and that disability began immediately after the blow. It may be true that the opinion was narrowly confined to physical examination regardless of history, but if it is true, it is easier to accept because the specialist was not a witness the scope of whose opinion could be freely explored at the hearing so that it could not be misunderstood. Perhaps also it is easier so to interpret the opinion because of a possible inference that the witness was sufficiently partial to his employer as to put on blinders that would confine him narrowly to his specialty. Finally, there was apparently no expert testimony to advise the court whether medical knowledge could help the court to make or refuse to make the connection between the blow and the disability, or whether so far as medical expertise is concerned this was an im-

¹⁷ The court did *Thoreau v. Industrial Accident Comm'n*, 120 Cal. App. 67, 7 P.2d 767 (1st Dist. 1932).

²⁸ 2 Wigmore on Evidence § 561 (3d ed. 1940).

ponderable, to be disposed of as an ordinary issue of fact (Logically, there are four possibilities: given these facts medical knowledge is (1) competent to infer that the disability was the exclusive product of the blow, (2) competent to infer that the disability was within certain limits of probability attributable to the blow within certain degrees of intensity of connection ("aggravating," "lighting up," "precipitating," etc.), (3) competent to infer a complete absence of relationship to the blow, or (4) incompetent to add anything to common experience that would be significant) If the opinion had been regarded as founded on all the believable evidence, it would, of course, have carried with it advice from the expert by implication that the court needed expert advice on the merits. The position of the court in drawing an inference of connection carries with it an implied holding that expert advice was dispensable, a doubtful conclusion even if there had been no expert opinion in the case. But the point is that it is unsatisfactory to leave it initially to the party having the burden of proof and ultimately to the tribunal to decide without expert assistance whether expert opinion is indispensable.

Also, it is unsatisfactory to rely upon partisan experts both for advice that they are needed and for advice on the merits. A specialist who does not believe his advice will be helpful and cannot be so persuaded is not likely to be offered. If an expert is offered, then, one result of his presence in the proceeding is likely to be an increase in the burden of proof, and the injured workman or his beneficiary is more likely to need an expert witness than if the employer or carrier had not offered one. Moreover, with the multiplication of partisan experts, the probabilities diminish that any expert will be found to advise against the usefulness of expert advice on the merits, even in a case that may appear to be close to common experience, or at the other extreme appears from the jangling contradictions of the supposed experts to be beyond specialized

knowledge. "Isn't there an old rule which suggests that a question never be asked to which the answer is not known?"¹⁸ Answer. The answer to this one is not known, unless someone discovers its existence; proof of non-existence of a rule is hard to develop, when there are no spatial or temporal limits to the search, and the conceptual limits are vague. However, it may be confidently asserted that enlightenment consisting in a report that the answer is not known may be most helpful to a tribunal faced with the problem of giving an answer one way or the other. Many of the "technicalities" of litigation are concessions to the shortness of life.¹⁹

Three weaknesses of partisan selection of experts may be distinguished. One is the absence of special skill to evaluate claims of expertness. Another is the suspicion of bias. Finally, there is the improbability of disinterested advice as to the helpfulness of expert advice. Partisan legal advice is not subject to these objections because the judiciary, with its familiarity with law-ways, is able to estimate the need for help to evaluate the qualifications of the advocate, and to discount the partisan influence. Moreover, so far as the partisan influence is concerned, the method is designed to recognize simultaneously divergent views, whereas in matters of fact the scientific acceptance of general propositions as only provisional can hardly go so far as to contradict the basic assumption of an existing consistent external Nature. Hence the incongruity of a "battle of the experts" in which conflict of opinion evokes suspicion of bias or false pretensions of knowledge or both, and men of integrity and ability in their own fields are arrayed in false colors, many times to their pain and disgust.

¹⁸ Hammon, "Explosion Damage Cases. Insurance Procedure and Expert Witnesses," 43 *Am Bar Ass'n Jour* 135, 185 (1957).

¹⁹ "The conditions under which the tribunal is required to conduct its inquiries, the limits of time and of the means of investigation, are such as no scientist would tolerate" Morgan, "Suggested Remedy for Obstructions to Expert Testimony by Rules of Evidence," 10 *Univ of Chicago L. Rev* 285 (1943).

These difficulties can be overcome in part by specialization of workmen's compensation tribunals. Even if they are not recognized as having a degree of specialized knowledge that entitles them to a scope of official notice that contracts the area of controversy,⁷⁰ their specialized knowledge can be brought to play in other ways. By a process of self-education they can determine with greater precision than, say, a judge who less frequently deals with medical questions, not what the *going medical rules* are, but what help the sciences of medicine can give. By a process of acquiring familiarity with the reputations of medical practitioners and others who may be called as expert witnesses they can obtain insights for the evaluation of qualifications of expert witnesses. So long as their decisions are binding on judicial review in terms of the substantial evidence formula, it will not greatly matter, except to save time in the proceedings, whether the evaluation of qualifications is expressed in a decision on qualification of the proffered expert or in a decision on the weight of his evidence. The point is suggested by an observation of the court in *Giant Grip Co. v. Industrial Comm'n*.

Counsel for appellants remind us that counsel for the commission, contending for a different rule in the Merton case, *supra*, argued that to insist on such a requirement [the apportionment of disability among successive employers] was to

recessive accidents. It is clear from the present record that counsel underestimated the daring of doctors and the credulity of commissions.⁷¹

It has been justly observed that a doctor who has studied medicine and observed patients primarily in terms of therapeutics may not be qualified to say whether injury can produce a certain disease, but that if he goes to court to accommodate an old patient he

may be qualified as a thoroughgoing expert and find it hard to confess the limits of his knowledge.⁷² The provisional character of scientific findings and their constant revision under conditions of modern research render it unlikely that the best current knowledge will be widely diffused and more likely that it will be obtainable from highly specialized students of a narrow field. The problem is how the officers charged with administration of workmen's compensation may best draw upon these resources. In the solution of this problem the contribution of the general practitioner, who is more likely to have collected and recorded or remembered the information that depends upon direct perceptual knowledge informed by the medical disciplines,⁷³ must not be ignored or discounted. The narrow specialist called in at a later time cannot be expected to supply reliable information when gaps in information that should have been obtained earlier stretch out the chain of inferences he is asked to make.

ORGANIZATION

The realm of currently advancing knowledge can be made available by organizing the methods by which expert opinion is brought to bear upon information. The traditional method in court is by the use of the hypothetical question.⁷⁴ Among its advantages are the employment of experts who have no way of knowing the truth of the facts assumed in the hypothesis, with the consequent specialization of functions as between them and other witnesses, and the opportunity of obtaining opinions from a given expert upon alternative assumed conditions. Among its disadvantages are the

⁷⁰ Smith, "Scientific Proof and Relations of Law and Medicine," 10 *Univ. of Chicago L. Rev.* 242, 247 (1943).

⁷¹ Contrast the approach, framed presumably in terms of a lay witness oblivious to the possibilities of litigation or other need to recall, described as "Stimulation of W by the Event," in Cleary, "Evidence as a Problem in Communicating," 5 *Vand. L. Rev.* 277, 283-287 (1952).

⁷² Busch, "The Hypothetical Question," 3 *Jour. Public L.*, 550 (1954).

⁷³ *McCarthy v. Ind. Comm'n*, 194 Wis. 198, 215 N.W. 824 (1927).

⁷⁴ 271 Wn. 583, 74 N.W.2d, 182 (1956).

complex shape some hypothetical questions take and their extreme prolixity, or on the other hand the vagueness with which others are framed, with the result in both cases that it is difficult for the auditor to have in mind the exact state of facts to which the opinion is addressed, if indeed it is not difficult to appreciate that the expert thoroughly understands the question. For the courts, the Model Code of Evidence and the Model Expert Testimony Act have shown the trend of thinking in favor of greater flexibility in specification of the hypothesis, to avoid the confusion attendant upon prolix questions,²⁵ and the desirability of court-appointed experts.²⁶ The policy with respect to appointment of witnesses by the court seems to be to avoid the difficulties inherent in a rigorous exclusion of testimony of expert witnesses whose qualifications are doubtful or of whom bias is suspected, merely by obtaining more reliable testimony. This policy puts the tribunal in a position to discount to zero, if it seems advisable, the weight of the former.

The picture of the small group of lawyers who have harnessed up the small group of expert witnesses, whose role as they conceive it is to win a case,²⁷ is reflected in the experiment in the courts of New York and Bronx Counties, New York.²⁸ Some of the features of this plan are adaptable to, and have been

in essence employed in, the field of workmen's compensation. The New York Academy of Medicine and the New York County and Bronx Medical Societies co-operated to assemble a list of highly qualified doctors in the special fields the justices indicated were needed. Not only were they highly qualified but none of them had been prominently identified with either plaintiffs or defendants in personal injury cases. A court rule set up the procedure, which calls for consultation of the justice with counsel for the parties, after which he may order the case referred to an impartial expert. The expert examines and reports through a Medical Report Office, the clerk of which makes copies of the report available to the parties. The impartial expert is furnished with the medical records the parties have or can obtain as far in advance of examination as possible. After copies of the report are distributed, the pre-trial conference is resumed. In the event of litigation, the trial judge or either of the parties may call the impartial expert as a witness. Fees for the impartial expert for testifying and for examining have been set by him, subject to supervision of the judge, and paid from Project funds.

Compensation plans have utilized the services of staff physicians and of special panels of private practitioners. However, the degree of reliance upon specialists as impartial experts has not been very great.²⁹ A more thoroughgoing degree of such reliance may be built into the organization and structure of the decision-making itself, by the establishment of medical boards with power to find facts binding upon the commission generally charged with administration.³⁰

Two kinds of hazard are involved in this plan. One is the range of considerations indicated above³¹ having to do with the proper

²⁵ McCormick, "Some Observations upon the Opinion Rule and Expert Testimony," 23 *Texas L. Rev.* 109 (1945).

²⁶ *Ibid.*, Ladd, "Expert Testimony," 5 *Vand. L. Rev.* 414 (1952).

²⁷ "We would have been more favorably impressed with the testimony of the doctor had he not laid so much stress on the great number of 'cases' which he had 'won' in various courts as compared with the few which he had 'lost'. We have always entertained the view that doctors neither 'win' nor 'lose' cases in which they testify as experts." *Smith v. W. Horace Williams Co.*, 84 So 2d 223 (La. Ct. of App. 1956). The court, however, held that his failure to gain membership in the American Board of Orthopedic Surgery did not disqualify him as an expert in orthopedics, although it could be considered in weighing his opinion.

²⁸ Peck, "A Successful New Plan: Impartial Medical Testimony," 42 *Am. Bar Ass'n Jour.* 931 (1956); Botein, "The New York Medical Expert Testimony Project," 33 *Univ. of Detroit L. Jour.* 388 (1956); *Impartial Medical Testimony* (1956).

²⁹ Yerion, "Expert Medical Testimony in Compensation Proceedings," 2 *Law and Contemp. Problems* 476 (1935).

³⁰ For example, in Maryland the commission cannot reverse the findings of the medical board on medical questions if the findings are supported by "legally sufficient" evidence. *Big Savage Refractories Corp. v. Geary, Md.*, 121 A 2d 212 (1956).

³¹ See pp. 468-470, *supra*.

employment of personnel with medical qualifications and the problem of disentangling medical from legal opinions. These hazards may be minimized by the careful referral of issues to the medical board in such form that legal questions are excluded and the medical board is free to concentrate its attention upon medical questions, and by framing the findings in such form as to disclose any errors in legal reasoning that may inhere in medical findings or procedure before the medical board. Nevertheless, these hazards are inherent in such a plan, especially in view of the duty of the medical board to hear lay testimony (which as testimony is not strictly a matter of medical competence to hear, but rather of trial examiner, referee, or trial judge competence) and in view of the limited range of medical specialties that can feasibly be represented on a permanent medical board operating as an integral part of a public agency.

The other kind of hazard resembles one of the vices of partisan expert testimony. That is the tendency of a group of experts in industrial medicine always functioning in the same role (conducting medical litigation) to acquire a set of official views representing the institution. Although one of the goals for improving the decision of issues of medical fact is the development of greater consistency, this consistency is not so desirable as to justify a fixed attitude in the interpretation of evidence, whether the effects are liberal or conservative.

Both kinds of hazards are minimized greatly if the medical boards are constituted on an *ad hoc* basis, with due regard for matching the qualifications of the expert body summoned for consultation with the issues in the case referred. Moreover, the range of expertise from which the members of such boards are drawn may be greatly increased. The general agency administering the plan will need a medical staff to assist in evaluating the need for a special medical board and the qualifications that should be mustered for it. A feature of the New York court plan is worthy of incorporation into this process: staff advice should be supple-

mented by the development of panels by medical bodies outside the compensation agency, such as medical societies and schools.

Earlier discussion in this paper has relied upon a distinction between perceptual knowledge and inferential knowledge. This distinction, for purposes of medical board functions, may be elaborated to indicate how much these boards can rely upon their own knowledge and to what extent the knowledge must come in through an adversary hearing process. Inferences drawn from observation, especially from expert observation, depend upon general knowledge. For example, judgment of the speed of an automobile, if based upon precise measurement, depends upon such general mathematical knowledge as is incorporated in the testing mechanism and also that which is employed by the observer in reading the results. If it is based upon raw observation, the general knowledge applied by the observer comes from a complex of memories of past experience. Much depends upon the skill, as well as the knowledge, of the observer in collecting the perceptual information. To this information, general or abstract knowledge is applied to produce a judgment. If the measurement mechanism is simple enough that little skill is required to apply it, the mechanism may record data from which a nonobserver can draw a conclusion. In drawing the conclusion the general mathematical knowledge that is built into the mechanism and that is employed by the nonobserver can be reliably applied to reach a conclusion. Another analogy is the hypothetical question itself, by which perceptual knowledge is verbalized and summarized for a nonobserving expert. The expert then purports to have only generalized knowledge from which to make an application to the knowledge supplied him. His contribution consists in adducing the generalized knowledge and applying it to draw a conclusion by inference.²²

²² Cf. Davis, "An Approach to Problems of Evidence in the Administrative Process," 55 *Harv. L. Rev.* 364 (1942); Jaffe, "Administrative Procedure Re-Examined: The Benjamin Report," 56 *id.* 704 (1943); 2 *Larson's Workmen's Compensation Law* 299-308 (1952).

Expert medical observation cannot be supplied by the medical board in its capacity as trier of medical issues.³³ For such information it must hear witnesses, lay and medical, and in general depart from its medical specialties into the legal business of according a fair hearing. Indeed, it is sometimes doubtful whether it can apply its own generalized knowledge without having expert testimony confirming it. At least its knowledge should be made apparent in the record, by appropriate findings, and preferably also by notice to the parties so that there is opportunity for issue and rebuttal at the administrative level.³⁴

The difficulty of verbalizing some kinds of perceptual medical knowledge (e.g., distorted facial expression, sweaty and flushed), the existence of nonmedical duties in the conduct of a trial, and the cumbersomeness of getting generalized knowledge in the record and recognized on judicial review as such and as a legitimate subject of official notice by a medical board—all these considerations argue against the use of a medical board. In its favor is its solution of the problem of choice between conflicting expert opinions. In its absence the courts of necessity swallow the spectacle of essentially uninformed choice by laymen between experts who are supposed to be informed but who disagree.³⁵

An alternative solution lies along the lines of the New York plan. Its tactic is to meet the problem of choice among conflicting expert opinions by providing a basis for choice of the impartial opinion as against an opinion suspected of partiality, or choice of the more authoritative opinion, whether from an expert chosen impartially or by a party,

as against the opinion of an expert whose expertise is suspected of shortcomings on the kind of issue involved. At the same time it affords full opportunity for issue and rebuttal and full freedom of choice. It goes further than partisan selection in giving the tribunal on its own motion an opportunity to seek a remedy for apparent gaps in expert opinion, and provides an impartial source of knowledge about the need for and the prospects of finding assistance from expert knowledge. It attempts to solve the problem posed by the limited diffusion of the best current knowledge in a narrow field.

The context of medical issues arising in a scheme of noncourt administration of workmen's compensation affords opportunities for adaptation of the feature of pretrial conference in the New York plan. Administrative processes can be so shaped as to obtain the impartial opinion at an early stage of controversy, well before hearing, so as to reduce the number of cases that have to be subjected to formal litigation. At the same time accommodations can be made that will reduce waste of the time of the expert, by appropriate scheduling. In some jurisdictions the opinion may be obtained in writing, and the analogue of the hypothetical question can be proposed in written form adapted to the needs of the case and influenced by conference among the parties and the tribunal, so as to avoid the awkwardness of propounding a verbose question orally. Thus the expert opinion can be expounded in a more deliberate and enlightening fashion. These suggestions seem to be applicable in large part also to experts retained by the parties.

Answers to the analogue of the hypothetical question should be more than naked conclusions.³⁶ They should elucidate the medical reasoning, and thus supplement the criteria for choice of opinion by relative impartiality and by relative record and reputation for expertise. They would then become the foundation for tribunal findings in the event of litigation, and these findings should then

³³ *Carstens v. Pillsbury*, 172 Cal 572, 158 P. 218 (1916).

³⁴ *Merton Lumber Co. v. Ind. Comm'n.*, 260 Wis. 109, 50 N.W.2d 42 (1951), and cf. 60 Stat. 241-2 (1946), 5 U.S.C. § 1006(d) (1952). *Davis*, "Official Notice," 62 *Harv. L. Rev.* 537 (1949).

³⁵ "The court, in going beyond the medical evidence in reaching an equitable decision, is not doing so on the supposition that it wishes to substitute its opinion for those who are specialists in that field, but it must do so when the evidence is confusing and lacking." *Hebert v. Fifteen Oil Co.*, 46 So.2d 328 (La. Ct. of App. 1950).

³⁶ *Osborn*, "Reasons and Reasoning in Expert Testimony," 2 *Law and Contemp. Problems* 488 (1935).

go a long way in giving the court on judicial review an intelligible basis for discerning the foundation in medical fact on which the order of the agency is based.

CONCLUSION

The division of functions proposed here asks the treating doctor to expand his investigation beyond the bounds of seeking knowledge for the purpose of healing, even into areas that are, strictly speaking, non-medical. The argument for this emphasis is the necessity of getting perishable information while it is still alive, and the strategic position of the treating doctor. The pro-

posals further suggest ways in which different kinds of medical expertness can be mobilized for their appropriate uses, so that the possessor of abstract knowledge in a narrow specialty can relieve other medical experts from the imposition of answering questions that others are better qualified to answer. Attention has also been given to some of the areas, such as the conduct of an adversary hearing, where the specialized skills and knowledge are legal rather than medical. A proper division of labor provides hope for the improvement of a complex of activities that purports to be rational and to employ the skills and knowledge of professional learning.

CHAPTER 54 RELATIONSHIP OF EMPLOYMENT TO THE DEVELOPMENT OF CARDIAC PATHOLOGY

R. G. Knutson

Perhaps all administrators of workmen's compensation laws have their respective problems with regard to determining whether liability might attach in connection with a heart attack that occurs while the individual is engaged in the performance of his or her duties. Some of these attacks occur while the individual is engaged in the performance of services and still others with variable degrees of elapsed time following employment. The administrative trend relating to handling of these claims varies materially in the respective states. In the majority of cases attempts are made to establish causal relationship between cardiac involvement and so-called accidental injuries, as distinguished from occupational injuries.

I am certain that most, if not all, administrators become confused at times with respect to attempting to make valid decisions regarding these claims. As a rule, the issues involve a finding of fact as distinguished from an issue of law. Further complications

arise with respect to securing a true history of what the involved individual was actually doing at the time and the symptoms immediately following the incident. This, of course, applies only to the nonfatal cases. Oftentimes the history is distorted by both lay and medical testimony.

I should like to proceed on the assumption and basis that all administrators should attempt to ascertain the facts regarding the alleged cause bringing about the attack and the matter of causal relationship. In their attempts to do justice, the administrators are dependent to a large degree upon medical guidance and opinions. It is unfortunate that on altogether too many occasions it becomes difficult to place proper value on medical evidence. This occurs particularly when apparently equally qualified medical men take opposite views and positions in connection with the interests of the respective parties to the dispute.

The foregoing has been said without any

intent or desire in any way to reflect upon the members of the medical profession. It is recognized fully that in some of these instances the circumstances may be such that honest differences of opinion may prevail. On the other hand, there are occasions when it is rather difficult, if not impossible, to reconcile oneself to the opposite views and opinions expressed by medical witnesses. Perhaps the greatest assistance to administrators of workmen's compensation laws could come from such a conference as this, by securing rather unanimous agreement regarding the setting up of essential guardposts for administrators. I am certain that the writer heretofore and his associates would be deeply grateful for such assistance.

An effort will be made to set forth some items that may be helpful in attempting to secure enlightenment on some of the present perplexing problems.

- 1 What are the essential elements to have in mind where there is a history of prior cardiac attacks as distinguished from those cases with no history of pre-existing pathology?
- 2 What consideration, if any, should be given to the age factor? These claims arise with individuals ranging anywhere from 30 to 65 or more years of age.
- 3 Name the essential symptoms to be sought for in determining when the attack took place. To what extent can reliance be had upon lay testimony in securing proper history of the symptoms? At times the history of these symptoms can be obtained from the stricken individual and on other occasions reliance must be had upon some other lay person who may have been present at the time of the attack.
- 4 What are the essential symptoms and findings to be obtained from the examination by a physician or physicians?
- 5 Where it is contended that a cardiac

attack is related to employment, what is the longest period of time following the alleged incident before recognizable symptoms should manifest themselves and at the same time either establish or eliminate causal relationship? There are cases where alleged symptoms appear immediately, and others where the symptoms do not manifest themselves until several hours after.

- 6 Administrators are often confronted with alleged causal relationship between cardiac pathology and employment where excess strain, stress, or effort has resulted. What enlightenment can be given as to what represents *excess strain or effort*?
- 7 We all know that different types of employment involve variable degrees of either strain or effort. Some individuals may have been involved in the same work for periods of months or years, and others may experience difficulty after working a part of a day up to several days on a new job when they have an attack. At times it is difficult to determine with reasonable definiteness whether the effort may have been in excess of that normally experienced, and if so, the probable amount of increased effort.
- 8 What type of accidental injuries to the chest may be considered as contributing elements toward the subsequent development of cardiac pathology? What is considered a reasonable period of time following direct trauma to the chest before cardiac symptoms should manifest themselves when causal relationship is alleged? In cases of fatal results what pathological findings are considered material to establish causal relationship?

Answers to the foregoing questions are usually susceptible to cross examination in attempting to establish the logic of the doctor's or expert's opinion.

CHAPTER 55 THE UTAH MEDICAL PANEL SYSTEM

Otto A. Wiesley

INTRODUCTION

The knowledge and experience gained through eleven years of practice as a lawyer before The Industrial Commission of Utah and fourteen years as Commissioner and hearing referee convinced the writer that some real effort should be exerted to eliminate the hit-or-miss methods of administering justice and to bring order out of confusion and chaos.

A detailed study revealed that, except for a few cases of Judicial legislation, the Supreme Court of Utah has been consistent in its application of the law to the facts. It has consistently held to the *unusual exertion* theory in heart cases. In a few cases the result might have been different had the facts been better presented to the Commission and the Supreme Court. However, the confusion that had developed is largely chargeable to the medical testimony. Our records are replete with medical testimony, recently described by Jerome Pollock as "unedifying and adversary-oriented." Often, testimony

clearly showed that the witness was either not qualified, had failed to study the medical issue, or tailored his testimony, knowingly, to aid the cause of the party who called him as a witness. For example, one medical doctor testified that the applicant had swallowed pyorrhea-infected blood, which caused a peptic ulcer in a normal stomach. When called before the Board of Censors, he stated that he so testified because the applicant lived in a trailer camp. Not all the unedifying adversary-oriented testimony came from quacks and unscrupulous medical men who were primarily interested in witness fees or who were influenced by the equally obnoxious members of the legal profession.

The enactment of the Utah Occupational Disease Disability Act in 1941 emphasized the need for a new approach. Very few medical doctors were qualified to diagnose silicosis or other chest diseases caused by exposure to dust. Many cases were denied for lack of medical evidence. In 1949, labor and industry joined with the Commission to correct

finding investigation by means other than formal hearing that is foreign to our traditions of administrative law or offensive to our notions of fair play. Within the authority of the statute, the Commission can make certain, without any expense to either party, that the disease condition of the claimant is fully explored, and that the findings presented are the most reliable medical science can afford.

MECHANISM AND RESULTS OF THE UTAH PLAN

Statistics of the first seven years of operation under the panel system are amazing. Nearly 200 panel decisions have been issued from which there have been only four appeals. In each appeal, the decision was affirmed. In only one case was the validity of any medical finding by the panel attacked. On the strength of the success of the panel experiment in the limited field of the initial experiment, the Utah Legislature extended the scope of the program by amendment to the Workmen's Compensation Act in 1955. As the law now reads, the Commission uses the panel method in all occupational disease and workmen's compensation cases if liability is denied by the employer and there are important medical issues that cannot be resolved by the procedures formerly available, short of a formal hearing.

The statutes specify that panel members must be specialists in the field of inquiry. Accordingly, the Commission makes its selections of panel members from lists submitted by the Utah State Medical Association which, incidentally, has always been in sympathy with the program. Panel members are paid directly by the Commission. In practice, it has been found expedient to designate a chairman of each kind of panel, who serves indefinitely. The other two positions on the panel are rotated among the specialists on the list provided by the medical association. This kind of organization permits the development of expertise, assures a desirable uniformity of approach to the problems, and allows expression to new points of view.

When the panel is formally constituted, the Commission collects all available information and submits its entire file to the panel. The order appointing the panel authorizes it to make such studies as it considers necessary, including autopsies in death cases, at the Commission's expense. A digest of the laboratory and clinical findings and other pertinent information from the files is prepared by the chairman and submitted to the members. After each member has studied the digest, the panel meets with the Commission Chairman, studies and discusses the medical problems involved, schedules any desired additional investigative procedures, and eventually makes its findings. Formal findings and conclusions are prepared by the chairman of the panel, and these are mailed to the parties in accordance with the statute.

Adoption of a panel system is certainly in harmony with the general compensation philosophy. It assures an equality of treatment scarcely possible where the litigants are required to make their own medical investigations and their own presentations of the results. On the question of whether truth can best be transfixed in the quiet atmosphere of the council chamber or in the frenetic atmosphere of the courtroom, much can be said. There is little doubt about the feeling of physicians on the subject. The American Heart Association, The American College of Physicians, and the American Medical Association have all indicated approval of a panel system. The sounds of courtroom battles on medical questions are neither musical nor edifying.

The expenses incident to the employment of the panel system are defrayed out of a special fund into which employers pay when one of their employees, who has no dependents within the meaning of the compensation act, is fatally injured by accident arising out of his employment. This fund is known as the *second injury fund* and it has a counterpart in most compensation acts. The Utah fund had reached such proportions before panel costs were imposed upon it that it has, so far, been adequate to support the program without appreciable dun-

ination. This is true even though the panel has not hesitated to consult recognized authorities inside or outside the United States in difficult cases.

The use of a medical panel of the kind described here is certain to promote a closer co-operation between the administrators of the compensation program and the medical association. Good medicine is an absolute essential to good administration.

THE UTAH PLAN AND CORONARY ARTERY DISEASE

The Utah Commission, by reason of good relations with the medical fraternity, has made a signal contribution to the solution of a problem which I am sure has vexed compensation administrators throughout the country. The problem is what to do about cardiacs. Although coronary occlusion is certainly the result of a well-advanced disease process, the possible accelerating effect of stress and strain in employment has been the justification for awards under compensation acts throughout the country. A major difficulty in treating cases of coronary artery disease under compensation acts is in determining the extent of disability. There is a tenacious feeling among employers, and the general public for that matter, that a person with coronary artery disease is, for all intents and purposes, unemployable. Even though an applicant for employment with a history of coronary disease may work to retirement without recurrence of disabling symptoms, employers are reluctant to take the chance that they may have a fatal claim on their hands.

The fairness of the Utah plan is attested by the fact that the reports of the panel are accepted by all parties without appeal, though the plan in no way obstructs the opportunity to appeal. Many sufferers from heart attack have been returned to gainful employment. Disability ratings in nonfatal cases have reached some degree of consistency and reasonableness. No longer are coronaries arbitrarily, ignorantly, and prejudicially rated permanently and totally disabled. We think,

justifiably, that there is a silver lining on the clouds of doubt, suspicion, bias and ignorance that have blurred the vision of justice in the past.

Careful analysis reveals that the Utah plan embraces these cardinal principles:

1. *The relationship of industrial effort to heart attack can only be determined by recourse to expert medical opinion.* Each heart claim is reviewed and evaluated by a panel of physicians.
2. *Medical opinion must utilize the most advanced knowledge in the cardiac field.* The Utah Heart Association and the Utah Society of Internal Medicine, utilizing the latest and most advanced knowledge within the field, have established medical criteria regarding the relationship between industrial employment and heart disease. (These criteria must be re-examined from time to time with the advance of medical knowledge.)
3. *Medical opinion must be expert.* The Utah panels are composed of physicians who are experts in the field of heart diseases, and are best able to understand the established criteria and evaluate the latest and most advanced studies in the field.
4. *Medical opinion must be based upon complete data in each case.* The medical panel examines the workman, if living, if dead, an autopsy report is made available. The panel is supplied a complete medical history as well as a complete account of the workman's activity occurring before, during, and after his work on the job. If the data are too fragmentary, additional information is obtained.
5. *Medical opinion must be unbiased.* The members of the panel must not be in the position of being either for or against the workman or the employer.
6. *Medical opinion is obtained before controversy develops.* The opinion of the panel is obtained at the administrative level when the claim is first filed, before definitive administrative action and prior to adversary hearing.
7. *Medical opinion is given to all parties affected by it.* The Utah Commission

transmits copies of the written report to the workman or his beneficiary, the employer, and the insurance carrier.

- 8 *Medical opinion does not deny due process of law.* Any interested party not concurring in the panel's report may demand a hearing. If a hearing is demanded, the Utah Commission calls the panel members as witnesses; their testimony is taken under oath and all interested parties are afforded the right of cross examination.

SUMMARY

The panel system has worked in Utah. It can scarcely fail to appeal to an objective observer as a better way to arrive at the medical facts than is the examination of

medical witnesses before a hearing officer who has had no formal medical training, but who has the responsibility to make the findings. Furthermore, the system is practically proof against the evil of purchased medical testimony, and it relieves the physician of any responsibility he may feel to be an advocate. As of April 1, 1957, fifty workmen's compensation cases involving difficult medical issues have been submitted to medical panels. No formal hearings have been requested. We are convinced that there are fewer denials because of the panel system. This all sounds like a long and complicated procedure. However, such is not the case. We dispose of cases faster by the panel method than by the old hearing method.

CHAPTER 56 THE UTAH PLAN FOR EVALUATING INDUSTRIAL CARDIAC CASES

Louis E. Viko

INTRODUCTION

The present paper considers the Utah plan for evaluating the medical data in cardiac compensation cases from the viewpoint of a cardiologist, now serving on panels under the present Utah plan, and having had many years of experience as an expert medical witness in cardiac cases before the Utah Industrial Commission and in state and federal courts. The discussion is not intended to be an argument for or a defense of the Utah plan but an attempt to evaluate its advantages and its possible weaknesses. It is left to others to draw any inferences regarding the practicability of this or a similar plan in other states.

This report will consider in some detail the method of operation of the panel in cardiac cases. Comparison will be made with the previous adversary-hearing procedure, not only to suggest the advantages of the panel system, but because the law providing

for the panels also provides for an appeal to a commission hearing and for further appeal to the Utah Supreme Court. To date, no case submitted to a panel for cardiac or other nonsurgical cases has been appealed to a commission hearing, but in the event of such appeal, procedure in the hearing should be corrected.

This paper will then consider types of cardiac cases considered by panels. To a limited extent, the discussion will include cases of the U.S. Employees Compensation Commission. It is worth noting that for many years that Commission has used a medical referee system whereby it submits a case to an appropriate specialist for the most complete examination possible (including hospitalization if necessary), and asks from the medical referee an unbiased opinion on the medical issues. Though the legal definition of accident is different, the medical issues are essentially the same as in Workman's Compensation.

EVOLUTION OF THE UTAH PLAN

The Utah plan for evaluating medical data in industrial cardiac cases evolved under the guidance of Mr Otto Wiesley, Chairman of the Utah Industrial Commission. He felt that the chief source of difficulty in the previous system lay in widely divergent medical opinion and testimony. Recognizing this difficulty, he sought the aid of the Utah Heart Association. In 1952, that organization joined with the Utah Society of Internal Medicine in a meeting of most of the specialists in cardiology and internal medicine in the state for discussion of the problem with Mr Wiesley. The discussion was confined to coronary heart disease as the major and most difficult problem. The widest divergence of opinion was expressed, and at length. Some of the conferees were adamant in the point of view that coronary occlusion and/or myocardial infarction is never related to accident, exertion, or strain. Others might have granted, with Masters, Dack, and Jaffe,¹ that 2 per cent of cases of coronary thrombosis occur during unusual exertion. Still others leaned toward the 32 per cent correlation of Yater *et al*² or the incidence of 35 per cent of fatal coronary sclerosis recorded by French and Dock³ to be present in those individuals dying one to several hours after vigorous exercise. Inevitably, some referred to the frequency of fatal coronary thrombosis during sleep. But Mr Wiesley pointed out that the Supreme Court of Utah and that of many other states had ruled repeatedly that some cases of cardiac death or disability were compensable under industrial accident compensation laws, and that Supreme Court rulings are ultimate facts of life. It then became apparent to the whole group that realistic, practical thinking could be devoted more profitably to means of determining in what cases a causal relation between industrial accident and heart disease was *probable*, and in what cases the relation was *nonexistent* or *improbable*.

During this discussion it was obvious that, even in a meeting of doctors, much time was wasted as a result of confusion of definitions

regarding the various aspects of coronary heart disease. Despairing of arriving at any practical solution in so large a meeting the group authorized the writer to appoint a committee to draft and submit recommendations to the Industrial Commission.

Deliberately, the writer appointed nine doctors representing the middle and extremes of the positions as expressed at that meeting. This committee met many times in the ensuing months with Commissioner Wiesley. In its early meetings, it again expressed the widest divergence of opinion but finally agreed that factual medical proof on the issues involved was not yet available and that, in the meantime, some practical criteria to *serve as a guide for medical testimony* and to the deliberations of the Commission, and perhaps of the Supreme Court, would serve in the interests of both the workman and his employer (and insurance carrier), and minimize the chaos of the previous system. To this end the committee proposed to the Commission a set of *criteria* and appended a set of definitions⁴ intended to minimize the confusion arising from confused terminology. Perhaps the most significant point of these *criteria* is that they represented, at least by delegation, a substantial agreement in principle by most of the specialists in internal medicine of the state. Since the appointment of the committee did not provide for a report back to its parent group, the report was mimeographed by the Utah Heart Association and mailed by the Industrial Commission to every doctor in the state. No protest resulted.

At least tacit approval of the legal profession was obtained on February 20, 1953, when Commissioner Wiesley secured the opportunity for the writer to present the issue to the legal profession of the state at an Institute on Personal Injury Litigation sponsored by the Utah State Bar Association and the University of Utah School of Law. In the discussion, no serious objection was proposed by the lawyers present. The address, which included the *criteria* mentioned above, was published in the official bulletin of the Utah State Bar Association.⁵

The point had now been reached at which the Industrial Commission and the medical and legal professions had, in some measure, accepted the idea that though the Supreme Court had made some cardiac cases compensable under industrial accident law, such causal relation was a very limited one and subject to some reasonable requirements of proof. Before and in hearings, the *criteria* served as *ground rules* but not by any means as rigid determining rules.

By 1953, Commissioner Wiesley had secured very effective operation of the medical panel system, authorized by law in 1949, for industrial disease disability evaluation. He had found it far superior to the previous adversary-hearing procedure, especially in handling silicosis cases. So in December, 1953, Commissioner Wiesley began submitting to medical panels cardiac cases under question under industrial accident compensation. As Goldwater pointed out in May, 1954,⁴ since there was no statutory provision for such panels, the legality of the action of the Commissioner in adopting a panel report may have been questioned. Clark⁵ commented that "there are probably few if any states where such a program could be enacted by law." However, in February, 1955, initiated by Commissioner Wiesley, and again with previous labor-industry agreement, the Utah legislature gave statutory approval, without controversy, for medical panels under industrial accident compensation law—not just cardiac, but all cases.

Some of the provisions of that law are important to the strength and permanency of the Utah plan in the event that the present chairman of the Commission should be replaced by a man of different caliber. The law provides: "where employer or the insurance carrier denies liability, the Commission shall refer the medical aspects of the case to a medical panel appointed by the Commission." Such reference to a panel is not left to the discretion of the Commission. The law also sets forth qualifications for the members of such panels. Other provisions are important to the thinking and procedure of the panels. "The medical panel

shall make such study, take such X-rays and perform such tests, including post-mortem examinations where authorized by the Commission, as it may determine." The wording of the law gives the panel wide latitude in securing all the facts but by implication makes it the duty of the panel to be diligent in securing such information. Of course, the law requires the panel to make a written report to the Commission. Such report is sent by the Commission to claimant, employer, and insurance carrier. If no objection is filed within fifteen days by the Commission, "the report is deemed admitted as evidence and the Commission may base its findings and opinion" on it but is not bound by it, if there is other, substantially conflicting testimony. The provision of greatest importance to the thinking and procedure of a panel is that, if objection is made to the report, the Commission is required to set a hearing. At such hearing the panel report shall be an exhibit but evidence only as far as it is substantiated by evidence submitted. Furthermore, any or all of the panel members may be required to appear at the hearing and be subject to cross-examination. Hence panel procedure, report, and opinion must be in such form that they can be justified in such a hearing or, in the event that substantially conflicting testimony is presented at that hearing, the panel may, without embarrassment, modify or even *retract* its opinion.

Panel procedure should attempt to retain the advantages of the previous *adversary* hearings and avoid the faults *therein*. The chief advantage of the hearing *up to the* free opportunity it gave to *each* present medical evidence, and *its* subject to clarification by cross-examination. A great deal has been written and said about the absurdities and inequities of *this* hearing, which Professor Ivan C. *Waller* has termed "medical-legal tournaments." *Some* criticism of such hearing procedure, as advanced by the writer, is included *here* to suggest errors to be avoided in *panel* procedure *and* even more to urge that *panel* reports *be* appealed to *Commission* *hearings*, *the* *best*

cedure in such appeal hearings should be very different from that followed before the inauguration of the panel system. Otherwise they could negate very largely the value of medical panels.

THE PREVIOUS INDUSTRIAL COMMISSION HEARING SYSTEM

Because I have confined my medical practice almost entirely to cardiology for the last thirty years, I have been called frequently to appear as a medical expert before industrial commission hearings, considering cardiac cases in relation to industrial accident, and I became increasingly convinced that the whole procedure was all too often a battle of wits between attorneys and medical witnesses rather than a sober inquiry into truth and justice.

Before the hearing the file of the Industrial Commission contained some medical reports of the attending physicians, usually adequate regarding the nature of the original injury and any surgical results thereof, but usually far from adequate regarding any alleged cardiac effects. If the Commission made, on its own behalf, inquiry designed to clarify the latter issue, there was no mechanism for such inquiry to be made by a competent independent physician for the Commission.

At the hearing the attending physician was expected to present the medical facts but seldom in a sequential, complete order, withholding opinion until after the establishment of all the facts. Instead, he had met first with the attorney for the plaintiff, who had tried to condition the attending physician to present the facts in the light most favorable to the plaintiff. To insure this the attorney for the plaintiff preferred that the medical facts come out in response to the attorney's questions rather than in narrative form. The Commissioner conducting the hearing might or might not permit this procedure, tending, as it did, to emphasize some medical facts at the expense of others of possible contrary significance. Then, in response to questions from the plaintiff's attorney, the attending

physician was expected to express opinions favorable to the plaintiff's claim for compensation for the alleged causation or aggravation of cardiac disease. No matter how honest the attending physician, he found it difficult to escape bias, owing to his personal relation with the patient or sympathy for the widow.

Since if left at this point all the advantage would lie with the plaintiff, it became necessary for the insurance carrier to present a medical expert to controvert the opinion of the attending physician and, lest this tip the scale too far in the other direction, the plaintiff now needed a medical expert. Someone has defined a medical expert as one who talks about a simple thing in so confused a manner that everyone is confused. With the help of the attorneys, this was frequently the end state the Industrial Commission was expected to resolve by some legerdemain.

No matter how great the integrity of the medical expert, he too came to the hearing conditioned as a witness for one party to the proceedings. He had been approached previously by an attorney or claims adjuster and had been furnished some of the factual data in the case—partial because it contained the often meager data in the files plus other, often partisan, data the attorney had been able to gather; obviously, it did not contain any new information obtained by the other attorney. It would be legal heresy to disclose information to the other party. If the medical expert could express the desired opinion upon such partial data, he became a witness at the hearing. An honest expert would have it understood before the hearing that, if new facts came out at the hearing, he reserved the right to modify or even reverse his opinion. Perhaps a more reliable witness would be found in his place.

Again, at the hearing the medical expert was rarely furnished first all the medical facts and then permitted to express an ordered, logical opinion without interruption. Rather, he faced a series of hypothetical questions from each side intended to obtain desired answers. Then the attorneys attempted to discredit and confuse the wit-

ness into contradictions. One result of this was to make the most honest witness more partisan.

To further the design that the medical witness should not have all the facts, the medical expert often did not hear new verbal testimony brought out at the hearing. Rather he heard only that part of it which another attorney chose to bring out in his questions.

In the whole matter of medical testimony as it was usually brought out, there was frequently an inequity in favor of the insurance carrier. Such carrier was always ably represented by a skillful lawyer and/or claims adjustor; all too frequently the plaintiff, if represented at all by legal talent, was represented by one less skillful and one willing to accept a contingent fee frowned upon by the more established members of the profession. The writer, appearing for the insurance carrier, in justice not infrequently felt forced to act in part as a witness for the plaintiff. The foregoing, implying the financial inability of the plaintiff to employ skillful legal representation, brings up the question of fees for medical experts. The insurance carrier was able and willing to pay an adequate fee for the time spent by the physician in preparation and in the hearing. The plaintiff often could pay no fee at all unless the decision was favorable to him. As far as the insurance carrier was concerned, an ethical physician made his fee dependent upon his time involved, regardless of outcome, yet unavoidably, if he appeared for the plaintiff, any fee at all might be contingent on the outcome. Perhaps this also gave an advantage to the defense.

Another source of confusion, and part of the reason the medical expert served so effectively to confuse, lay in the absence, especially in cardiology, of commonly acceptable, simply defined terminology.

The greatest source of confusion lay in the fact that our scientific knowledge of the role injury, exertion, strain, and emotion play in the causation or aggravation of heart disease is far too scanty. Hence there was the frequent spectacle of equally competent experts

giving opposite opinions even on the same set of facts. Free use of the words *possible* and *probable* in relation to causation did not help.

THE MECHANICS OF THE UTAH MEDICAL PANEL SYSTEM

In the adjudication of its cases, the Utah Industrial Commission uses many types of panels: silicosis, surgical, orthopedic, ophthalmologic, etc. Here we consider only the operation of panels that consider cases involving illnesses generally covered by the specialty of internal medicine, including the subspecialty of cardiovascular disease. For convenience in discussion here, these are termed *medical panels* in contradistinction to surgical and other panels.

Both strength and weakness are found in the operation of our *medical panels*. Each panel is selected by Commissioner Wiesley from a list of specialists certified by the Utah State Medical Association. In the interest of economy of State funds and the time of the panel physicians, three cases are assigned to a given panel at about the same time. Except for the Chairman, a new panel is assigned for each set of cases. In the opinion of the writer, the Chairman should now be rotated. The Industrial Commission sends its complete original file of the case to the Chairman of the panel, who reviews it and takes steps to secure such additional factual information as may seem needed. Ordinarily, if the medical data from attending physicians is incomplete, the panel chairman writes to them for additional information, referring to the fact that the panel is acting for the Commission. Up to the present time, every physician has been entirely co-operative. Some have gone to considerable length to furnish detailed and lengthy reports. Few have been argumentative or partisan in such reports. It is our experience that because they are writing to physicians, they are much more hesitant to draw extreme conclusions than in the frenetic environment of a hearing. If any physician failed or was unwilling to furnish requested information, Commissioner Wiesley

could make a direct request or order. In every case, with permission of the attending physician, hospital records are reviewed and summarized. Occasionally a hospital record is brought to the later meeting of the panel. So far, it has not been necessary to use the Commission's power to subpoena a hospital record. More recently, we have made it a practice to write to the plaintiff or his attorney, if he has one, requesting any information either cares to submit. Less frequently, we have made similar requests of the insurance carrier or its attorney. If non-medical factual data such as the circumstances of the accident need amplification, such data are usually secured for the panel by the Commission. In death cases, an autopsy report is usually available, since the Commission is authorized by law to require a post-mortem examination, even if it necessitates exhumation.

When the Chairman of the panel considers the data as complete as possible, he summarizes all the data in multiple copies, identifying the sources. The entire augmented file and summary are now sent, in turn, to each member of the panel for study at his leisure. He checks the summary against the file. He retains one copy of the summary. Any panel member may suggest that there is a need for securing additional data.

When each panel member has studied the file, the panel meets with Commissioner Wiesley to discuss the three cases. The summary may be amended. The panel then drafts a report, which includes the summary, with an opinion on the medical issues of each case. The report, when typed in multiple copies, is sent to each panel member for correction or signature and then forwarded to Commissioner Wiesley, each panel member retaining a copy for his personal file so that, if the case came to a hearing later and the panel member is questioned, he will know not only the opinion of the panel but the data upon which that opinion had been based.

The panels have not examined the plaintiff personally. In cardiovascular cases in which examinations with X-ray and electro-

cardiograms often are necessary, such examination by the panel would be impractical. When necessary, the Commission has referred the plaintiff to an independent internist or cardiologist for any necessary examination prior to the meeting of the panel, such examination being done at the expense of the Commission.

In some instances, we have had the attending physician meet with the panel for presentation of his observations of the case and to clarify the issues. Sometimes, but not always, we have had such a meeting transcribed by a court reporter.

There are some weaknesses in the operation of this panel system. It is possible that neither party gets as free opportunity to present the medical aspects of the case as in an open hearing. In the panel procedure, the correspondence and collection of information may take more time than if it were all collected at one time in a hearing. This may result in delay that may be a hardship to the plaintiff. Further time elapses between the time the panel chairman forwards the file to the other panel members and the time the report reaches the Commission.

A case considered recently by one of our medical panels illustrates the difficulty of securing complete medical data. It was thought that it might be the first case appealed to a Commission hearing. A covered workman suffered a compression fracture of the lumbar vertebra in July, 1955. The Industrial Commission file contained very satisfactory initial and continuing orthopedic reports with evidence of objective but not complete symptomatic recovery and return to light work for six months. In July, 1956, a report was submitted by an internist that during the previous month he had hospitalized the plaintiff with severe rheumatic heart disease, a duodenal ulcer, and epilepsy, as well as orthopedic complaints, possibly referable to the original injury. The report of this internist expressed the opinion that the symptoms of the plaintiff were directly related to his injury of a year before, but did not make it clear whether in asserting such relation to the injury he referred only to the orthopedic

complaints or to all the complaints. The file contained no further report from the internist except a report of death in October, 1956; no cause of death was given. The file contained an excellent autopsy report showing death due to decompensated old rheumatic heart disease with multiple peripheral and pulmonary emboli. An attorney for the widow claimed compensation upon orthopedic grounds from the original injury and did not refer to the heart disease. A letter from the panel to this attorney requested such information as he cared to submit. His reply enclosed a statement from the widow which admitted pre-existing heart disease but stated that both the orthopedist and the internist had told her that the injury had contributed to the injured workman's death. A letter from the panel to the orthopedist brought a reply that he did not feel competent to state what relation the back condition had to the heart disease. A letter from the panel to the internist brought little factual information but a vigorous opinion that the back injury was an "indirect, contributory and, indeed precipitating cause" of the death of the plaintiff. A further letter from the panel to this doctor asked for further justification of this opinion. A reply gave further factual information but did not meet directly the issues raised.

The panel chairman invited the doctor to meet with the panel. He accepted without hesitation, even though it actually involved a forty-mile trip to an evening meeting. A court reporter transcribed the meeting. The doctor was asked to present, in any way he chose, a chronologic review of his observations and treatment of the patient. This he did very freely. Without hesitation he brought out the fact that he had treated the patient for severe heart disease for many years prior to the accident and that prior to the accident the patient had congestive failure and one probable episode of embolism. Questioning by panel members led to a flat admission by the doctor that, "I cannot, and do not, say that there was any exacerbation of cardiovascular disease prior to June, 1956"—eleven months after the accident and six months

after the worker had returned to light work. The panel was satisfied that such frank and honest testimony and opinion by this doctor would not have been obtained in the old adversary hearing where the doctor was being heckled by two sets of lawyers and probably sat facing the widow and even some sad-looking children. In any case, the panel procedure seems to have been such in this case that it could face appeal to a hearing with reasonable assurance.

Yet in many cases difficulty in securing adequate medical information remains one of the weaknesses of the panel deliberations, but perhaps no more so than in the previous adversary hearing plan. Our panels need to do more than they have in the past in evaluating disability not just in terms of compensation, but in terms of rehabilitation and re-employment. Our panels are authorized to seek medical opinion anywhere in the country. In rare cases, a panel has submitted alternate opinions dependent upon later determination by the Commission of quasi-medical facts.

APPEAL FROM THE MEDICAL PANEL OPINION

As has been suggested above, either party may properly object to a panel report and secure a hearing before the Industrial Commission. Such hearing could easily have all the faults of the hearings prior to the panel system and could render panel procedure futile. It is, therefore, of the greatest importance that the procedure of such a hearing be designed properly. It should clearly separate legal from medical issues. Either party should be permitted to present previous or new medical data and these should be subject to contradiction or verification by either party. *The complete factual medical data should be presented before any medical opinion is asked.* Each medical witness of whom an opinion is to be asked should either sit through the whole presentation of factual medical data or have the opportunity to read it in transcript at leisure. Each medical witness should then be permitted to give, in his own way, an opinion on the medical issues

only, based upon *all the factual medical data*. Only then should he be submitted to cross-examination to amplify, clarify, or support his opinion. By this procedure it should be possible to eliminate the hypothetical question. Cross-examination should be designed to clarify and not to confuse or anger. A well-qualified commissioner conducting a hearing could and should give such direction to the proceedings. It seems possible that it might even be an improvement if some well-qualified physician, responsible only to the Commission, were to be delegated by the hearing commissioner to ask questions regarding medical fact and opinion, not expressing any opinion himself.

ACCIDENT, INJURY, TRAUMA, EFFORT, EXERTION, AND STRAIN IN RELATION TO CAUSATION OR AGGRAVATION OF HEART DISEASE

Whether the medical facts are well or poorly presented to the medical witness or panel member, he must be able to form an opinion whether or not a certain event or series of events either caused or aggravated heart disease. The cumbersome title of this discussion suggests the varied events that may or may not be supposed to have such relation to heart disease. Definitions add to the confusion. For example, Webster defines trauma not only as an injury or a wound, but as mental shock or the resulting condition from either of these. Again, Webster defines "injury" very broadly as damage or harm. Effort is defined as exertion or endeavor. Strain is given a multitude of definitions of which the most pertinent to our subject is "to stretch beyond its proper limit." Accident is defined as "something that happens unexpectedly or it is done unintentionally, usually a mishap," and a mishap is defined as "bad luck." Perhaps Webster may have helped to confuse legislators and judges. The various legislatures who wrote the original accident compensation laws probably were thinking largely in terms of wounds. Changing laws and court decisions have

broadened coverage until injury almost reaches the definition given for mishap, namely "bad luck."

It is not the duty nor prerogative of the doctor to determine whether or not a given event or series of events constitutes an injury for purposes of the state's industrial compensation law. It is his function to express an opinion whether an event caused heart disease or changed cardiac functional effectiveness, and to what degree, and for how long. If the law of a given state so prescribes, it may be the doctor's duty to decide whether such event aggravated a pre-existent heart disease or affected its course unfavorably and, again, to what degree and for how long.

Where external violence is involved—injury in its original and restricted sense—the role of the doctor is comparatively easy. Penetrating wounds of the heart present little difficulty to the medical witness. While non-penetrating injury to the chest may raise troublesome questions regarding that diagnostic entity, myocardial contusion, such problems are so infrequent that this discussion can be more profitably concerned with those cases where there has been no wound or injury of the chest, but where the event supposed to have affected a heart condition was exertion, effort, or strain.

Much as the physician would like to leave to lawyers, judges, and legislators the definition of such terms and confine himself to trying to relate an event to the heart, he too, in purely medical thinking, cannot escape making a distinction between exertion or effort and strain. Obviously, even in sleep, the heart makes an effort with each beat. The physician prescribes or limits some degree of effort or exercise for every heart case. He tries to persuade the heart patient to avoid strain, according to the Webster definition of strain, to avoid "stretching it (the heart) beyond its proper limit." The physician recognizes that this proper limit may be different for each patient. Practically, he assumes that whatever exertion the patient can usually carry on without signs or symptoms of diminished heart function is within such

proper limit. Thus usual exertion becomes proper exertion and conversely, unusual exertion may be strain as defined above. These concepts are commonly accepted medically. Even the doctor most adamant in denying any relation between exertion and coronary occlusion does not advise a known coronary disease case—or even any supposedly healthy sedentary man of 55 years—to engage in some strenuous activity to which he is not accustomed. There is, then, some validity for considering that the medical concept of heart strain has a bearing on the legal concept of unusual occupational exertion, if the latter be defined by law or court decision as an industrial accident.

To apply this point of view in the medical aspect of adjudication of industrial claims is not easy. Even where no insurance claim is involved and the physician knows the exact cardiac status of his patient, he often finds it difficult to prescribe the proper limits of exertion, occupational or nonoccupational. In insurance cases, the cardiac status of the employee at the time of the alleged injury or unusual exertion may be unknown, partly known, or only inferred from an autopsy. In addition, it may be difficult to determine the degree of exertion involved in the accident and in comparison with the employee's usual occupational or nonoccupational activity and one must assume, correctly or not, that such usual activity did not produce cardiac symptoms or signs and was, therefore, acceptable for that individual. It must be granted that the usual activities of an employee's occupation may be beyond the limits of tolerance of his heart and over a period of time adversely affect his heart. Fortunately for the medical witness, compensation laws do not yet consider employment alone a sufficient causative factor for heart disease under industrial accident compensation laws. The medical witness, then, is asked only to determine the adverse effect of some particular event (unusual exertion) or of a series of such events.

Excluding penetrating and nonpenetrating wounds of the chest and excluding the very rare case where extreme exertion injures a

normal heart, we deal with aggravation of a pre-existing heart condition, whether or not such heart condition was previously known to the employee.

Since perhaps 90 per cent of cardiac problems of employed workers fall into rheumatic, hypertensive, or arteriosclerotic etiology, the present discussion will be confined to these types. The rheumatic and hypertensive cases are somewhat simplified by the fact that chronic symptoms will have led many such workers to seek medical advice before an accident. Many will already have been restricted on medical examination, lessening the chance of cardiac strain, and in the event of an accident, there may be available data regarding the nature and degree of such heart disease. In the event that no such data are obtainable, examination or autopsy after the accident permits reasonable inference to what degree the natural course of the disease may have been changed by the alleged strain or accident. Nevertheless, in either case, it must be admitted that we cannot be too didactic about how much unusual exertion would be required and over how long a period of time it must have occurred to be sufficient to change, substantially and adversely, the natural course of either rheumatic or hypertensive heart disease. If every employed worker had been studied by cardiac work evaluation methods immediately before and immediately after the supposed accident, answers would be obvious. In view of the impracticability of this, the medical witness must usually rely upon the assumption that the usual occupational and non-occupational level of activity prior to the accident reasonably indicated the functional capacity of the worker at that time and that any substantial change in his ability to carry on such a level of activity indicates cardiac strain from the alleged unusual exertion, if such change occurred within a short time after the accident. Of course, the crux of the problem lies in definition of the word "short." It does not seem to me that any arbitrary limits can be set but, applying non-insurance experience, such time limit should

probably lie within the range of immediate to several days. If the latter limit seems unduly prolonged, one need only to recall that there may be an interval of several days between the onset of occult edema and pitting edema of the extremities. Fortunately, in the rheumatic and hypertensive cases, sudden death at work is so uncommon that causal relationship is more easily determined and insurance losses are less. Many workers with rheumatic or hypertensive heart disease are susceptible of rehabilitation and re-employment.

It is the arteriosclerotic cardiac group that creates the greatest problems in industrial insurance. Neither routine pre-employment examination nor much more careful examination by the employee's physician may detect coronary artery disease. A worker honestly free from cardiac symptoms may die suddenly of a coronary occlusion while on the job. If still living after the alleged accident, it may be very difficult to infer from the findings at examination what would have been the natural course of the disease. Even the degree of disability may be difficult to estimate, as in the case of the man with few or no findings, even in the electrocardiogram, but who complains of frequent angina, in which situation one must rely solely on the patient's statements.

Admitting that exertion, even overexertion, does not cause coronary arteriosclerosis and admitting that as in other types of heart disease if the disease is sufficiently advanced, overexertion may aggravate it to the point of congestive failure, we are still left with the question of whether or not unusual exertion can cause coronary thrombosis or myocardial infarction or both. There is a tendency to deny the former and accept the latter in some cases. Such distinction seems to the writer more academic than practical. There are too many noninsurance cases, the deer hunters and duck hunters, for example, usually sedentary businessmen of middle age, who die suddenly during sudden unaccustomed exertion, for me to accept such cases as pure coincidence. Post-mortem examinations usually show coronary thrombosis or

marked coronary arterial narrowing without myocardial infarction. It does not negate causal relation in these cases, even if one extended the usual statistics, that most cases of coronary thrombosis or infarction or both occur without unusual exertion, to limit the unusual exertion cases to 5 per cent or even 1 per cent.

If one admits that some cases, however few, of coronary thrombosis or myocardial infarction or both are precipitated by unusual exertion, we still have far too little knowledge of the time interval between such exertion and a recognizable attack. Do we know how long after the rupture of an atheromatous abscess complete thrombotic occlusion of a coronary vessel occurs? Do we know whether the pain we recognize clinically as the usual first symptom occurs during such thrombotic process or only after the development of a myocardial infarct? If the latter, how long does it take the infarct to develop sufficiently to give the pain? To what extent do different degrees of involvement of other coronary vessels or of collateral circulation modify the time interval? Clinically, one sees, at one extreme, a man who in anger ordered his son out of the house, had severe sudden pain as the son went out the door, and died of a coronary thrombosis. At the other extreme is a previously healthy man who, over a period of several days or weeks, develops angina on successively less exertion, then at rest, then during sleep, and finally has the characteristic prolonged pain of a proved coronary thrombosis and infarction. If unusual exertion was claimed as a causative factor in such a case, do we know when the coronary thrombotic process began? Do we know enough to deny an aggravating causal relation, unless such exertion occurred *during or immediately following* the strain?

Apropos of these questions, Clark⁷ cites a case of a man who not only had no cardiac symptoms but felt unusually well for a week before the onset of symptoms two days before death from rupture of a myocardial infarct, yet in whom microscopic examination of the infarct showed it to be at least seven to eight days old. Hence, the man had an

asymptomatic infarct for at least five days. If this man had had an unusual exertion eight days before death, would it have been proper to exclude an aggravating relation because symptoms were not immediate? Moritz⁸ notes that microscopic examination of myocardial infarcts almost invariably establishes the fact that the thrombus or the infarct or both had started to develop hours or days before death occurred and "could not have been caused by the terminal episode of stress." But on the same pathologic data, is it not possible that a stress hours to days before the onset of symptoms or sudden death could have had a causal relation to the beginning of the process of thrombosis or infarction?

The foregoing was the type of thinking that led the committee establishing our so-called *criteria* to postulate arbitrarily and by compromise the suggestion that "It seems reasonable to us to assume a probable relation between the exertion and coronary thrombosis with myocardial infarction, if new symptoms of cardiac disease appear shortly after that exertion and especially if such symptoms appear within one or two hours after such exertion—causal relation improbable, if symptoms delayed more than twelve hours." The same time interval was allowed in those cases of *sudden death* where there was coronary occlusion without infarction. Our panels have been influenced by these concepts but have not adhered to them completely. These time relations for stress and coronary thrombosis with or without infarction are more liberal than those proposed by Aronson for the State of Washington,¹⁰ which require that symptoms develop during or immediately following the exertion or strain.

SOME PRACTICAL EXPERIENCES WITH THE UTAH PANEL PLAN

The situation in Utah has discouraged the filing of any considerable number of extravagant claims. Hence, the number of cardiovascular compensation cases considered by our panels to date is far too few to treat

statistically. Rather, they are considered illustrative of types of medical problems reaching panels.

Omitting noncardiovascular cases and cases that are primarily vascular, the cardiac cases may be divided roughly into one fifth rheumatic heart disease, three fifths coronary heart disease, and the remaining one fifth other etiologic types of heart disease. The United States government cases occurred in a similar frequency.

The rheumatic group was not particularly difficult, if sufficiently complete medical data were obtained. One case has been described above in the discussion of the panel procedure.

The miscellaneous group illustrated two situations compensable under Utah law. One case was that of a worker who received steroid treatment for an industrial eye accident for a long period and who died suddenly. When the autopsy failed to show any cause of death, an opinion was obtained from Dr. George W. Thorn of Harvard Medical School. It was felt that death was probably due to the effect of an electrolyte imbalance upon the cardiac mechanism. In another case, an industrial hot asphalt burn of the foot in a known diabetic was followed by continuously recurring infection of that foot. Death occurred from hypertensive cardiovascular disease, the panel felt that there was a reasonable, continuous, causal chain from injury through infection to death.

The coronary artery disease group gave the greatest trouble. Even here, however, some cases were comparatively easy. For example, in one case the interval between the usual exertion and the first cardiac manifestation was eight months. In two of the Federal cases the strain was obviously so great that causal relation to coronary thrombosis with myocardial infarction seemed inescapable. Both employees engaged in severe and prolonged exertion at very high altitude in cold and stormy weather. It is of interest in connection with the question of time interval, that in one of these cases the first symptom appeared during exertion, and in the other symptoms were delayed about eight

hours, appearing as the man tried to get to sleep

In other cases, the alleged strain was obviously no greater than the usual occupational exertion of the employee. In these cases, the panels did not accept a causal relation medically. Regardless of the panel report, the Commission would no doubt have rejected the claims under the legal definitions of accident. In one case the worker was doing light manual work at the time of onset of symptoms of infarction but, shortly before, had been engaged in strenuous manual labor on his small farm. This case illustrates our point of view that the alleged strain should be compared not only with the usual occupational activity, but also with the usual non-occupational activity of the worker.

Moritz⁹ has suggested that in fatal cases the pathologist may be able to estimate the duration of the coronary thrombus or infarct or both closely enough to correlate the onset with the time of the alleged overexertion. However, two cases illustrated situations in which the pathologist could give little help. Both workers died suddenly while at their usual work. In both, the autopsies showed extreme coronary sclerosis with very marked narrowing of one or more arteries; neither showed either thrombosis or infarction. No other cause of death was found. Presumably both died of ventricular fibrillation. In both cases the issue was whether or not there was any cardiac strain (unusual exertion) involved in the job preceding death.

CONCLUSIONS

From the standpoint of the doctor, the Utah plan is far superior to the past procedure. Panel procedure needs to be improved, especially in securing more complete and detailed information. The mere inclusion of specialists on the panel does not insure an adequate impartial report unless such adequate data are actively sought out. The greatest difficulty in panel deliberation,

as in the past, lies in lack of scientific knowledge of the pathologic physiology of coronary disease and correlation of this with clinical manifestations, and second, the lack of a better understanding of the role of exertion and emotion in the evolution of the coronary disease process. It is hoped that this Conference will help to fill in some of these gaps.

BIBLIOGRAPHY

- 1 MASTER, A. M., DACK, S., and JAFFE, H. L.: Factors and events associated with onset of coronary artery thrombosis, *JAMA* 109 546, 1937.
- 2 YATER, W. M., WELSH, P. P., STAPLETON, J. F., and CLARK, M. L.: Comparison of clinical and pathologic aspects of coronary artery disease in men of various age groups, study of 950 autopsied cases from the Armed Forces Institute of Pathology, *Ann Int Med.* 34 352, 1951.
- 3 FRENCH, A. J., and DOCK, W.: Fatal coronary arteriosclerosis in young soldiers, *JAMA* 124 1233, 1944.
- 4 *Coronary Heart Disease and Utah Industrial Accident Compensation*. Report of a Committee to the Utah Industrial Commission, December, 1952.
- 5 VIKO, L. E.: Medico-legal problems of heart disease in relation to injury, *Utah Bar Bull.*, March-April, 1953.
- 6 GOLDWATER, L. J.: *Memorandum on Workmen's Compensation and Heart Disease with Particular Reference to the Utah Plan*. Report to the American Heart Association, September 7, 1954.
- 7 CLARK, R. J.: *Workmen's Compensation in Relation to Heart Disease*. Presentation to Congress on Industrial Health, Washington, D.C., January 25, 1955.
- 8 RUTLEDGE, I. C.: *Order Out of Chaos*. Monograph. Employment of the Cardiac, Washington State Heart Ass'n, September, 1955.
- 9 MORITZ, A. R.: Trauma, stress and coronary thrombosis, *JAMA* 156-1306, 1954.
- 10 ARONSON, S. F.: *Effects of Effort on the Diseased Heart*. Medicolegal implications. Monograph: Employment of the Cardiac, Washington State Heart Ass'n, September, 1955.

CHAPTER 57 DISABILITY DUE TO CARDIAC IMPAIRMENT UNDER THE DISABILITY INSURANCE PROGRAM

Arthur B. Price

During the period July, 1955, to August, 1956, 27.4 per cent (47,094) of the number of workers for whom a period of disability was allowed (172,158) were disabled by diseases of the circulatory system. Diseases of the circulatory system ranked first as a cause of disability as defined by the Disability Insurance Program. This experience is colored by the fact that one fourth of all persons allowed were over 65, by reason of the retroactive features of the program. Currently, the proportion of applicants 65 and over is very small and in our experience to date the incidence of cardiac disabilities among persons under 65 is approximately the same as for diseases of the nervous system and sense organs.

From the foregoing, the reason for the interest of the program in the proceedings of this conference is clearly evident. Other major causes of disability were: Diseases of the nervous system and sense organs including cardiovascular, 23.0 per cent; mental, psychoneurotic, and personality disorders,

13.3 per cent, infective and parasitic diseases including all types of tuberculosis, 12.0 per cent, and diseases of the respiratory system exclusive of pulmonary tuberculosis, 7.2 per cent. These diseases produced a total of 82.9 per cent of disability found in 172,158 workers who applied for benefits under the program.

The capacity of a cardiac case to perform any kind of substantial gainful activity is an evaluation that must be made in the determination of disability under the program. Perhaps the results of making such determinations on a substantial number of cases will in some way provide this group with information useful in answering the many questions provoked by the theme of this conference, namely, "Work and the Heart."

This presentation includes two types of information. The first part explains the program and is essential for the interpretation of statistical tables on experience of the program in evaluating disability resulting from diseases of the circulatory system.

BRIEF HISTORY OF DISABILITY PROVISIONS OF FEDERAL SOCIAL SECURITY PROGRAMS

Public Law 761, 83d Congress, amended Title II of the Social Security Act by including a provision designed to correct a hardship situation with respect to old-age and survivors insurance (OASI) contributors totally disabled for long periods during their normal working lifetime.

Prior to the enactment of what is now commonly called the disability "freeze" provision, OASI benefit rights could dwindle or could be lost entirely when an individual was prevented from continuing work by a totally disabling impairment before reaching retirement age, or prior to death. Under the old law the average monthly wage had to be figured from a specified starting date until the worker became 65 years of age or died. Those unable to work for a long period of time because of disability had their average earnings reduced by these periods of no earnings or of low earnings. These months of disability also had to be counted in figuring whether or not the person had done enough work covered by the law to make a payment possible.

Now, under this freeze provision, such non-work periods are excluded when figuring *average monthly earnings* and *insured status* of the totally incapacitated OASI contributor. Thus, benefits under social security will more nearly reflect actual earnings. A worker (enduring a period of prolonged total disability) can, through the operation of the freeze, preserve intact his benefit rights. This is in a sense, therefore, analogous to the *waiver of premium* provided in many commercial life insurance contracts.

Beginning with the month of July, 1957, disabled workers between 50 and 65 years of age who meet the work and disability requirements specified in the social security law may be paid monthly disability insurance benefits. The amount of the payments will be the same as the amount of the old-age insurance benefit for which the worker would be eligible at age 65 with the advantage of the provision for freezing his record of earn-

ings; that is, his benefit amount will be figured as though he had reached 65 on the date his disability began. A worker's dependents also receive any benefits based on his social security account while he is receiving his disability insurance benefits.

If a disabled worker is also entitled to a disability benefit under the program of another federal agency or under a state or federal workmen's compensation law, his social security disability benefit will be reduced by the amount of that other benefit. No disability insurance benefits under the social security law are payable for the first
though
if age or

Also, the 1956 amendments made it possible for a new group of dependents, disabled children 18 years of age and older, to receive monthly benefits based on the earnings of a retired or deceased parent. Up until January 1, 1957, a child's old-age and survivors insurance benefits have stopped when he reached 18, whether or not he could ever be expected to become self-supporting. The children now eligible to receive benefits may be of any age if they are unmarried, became disabled before they were 18, and have remained totally disabled ever since. To qualify for these benefits, a child 18 or over must be determined to be totally disabled for work. The same criteria for determining disability and the same evidence requirements apply in the case of a disabled child as in the case of a disabled worker who is applying for disability insurance benefits or to have his social security record frozen. However, the child need not have any record of work under social security. The mother of a disabled child may also qualify for benefits of her own, if the child is in her care.

GUIDELINES DEVELOPED AS ADJUDICATIVE METHOD

In any discussion of disability, it is essential first to define our terms. The old-age and survivors insurance disability provisions define disability as "Inability to engage in any

substantial gainful activity by reason of any physically determinable physical or mental impairment which can be expected to result either in death or to be of long-continued and indefinite duration. . . ."

Under the definition prescribed in the law in concrete terms, still, as in most statutes, it leaves us the responsibility for working out the application through administrative interpretation. Criteria had to be devised to answer such obvious questions as, "What is meant by 'continued and indefinite duration'?" Is the term to be used in the sense of *absolute continuity*, *eternal*, or are we to interpret the phrase to mean merely *not temporary* or *not transient*?

The concept of lack of capacity for "substantial gainful activity" is, of course, the difficult element in the definition. In applying the definition to actual cases, we do not construe it to mean that complete and incontestable helplessness must be demonstrated. However, congressional deliberation indicates that the definition was intended to mean *total*, in the sense that it refers to inability to engage in any substantial work, not merely the kind of work the applicant is engaged in, or the kind for which he is not obviously suited. Thus, an individual who has been advised to give up his particular kind of work in order to make his medical treatment more effective, or who finds he is no longer able to meet the physical and mental demands of the job, may not necessarily be disabled under the old-age and survivors insurance definition of disability. This might be because the impairment, although disabling for the person's usual occupation, cannot be considered disabling for all substantial gainful activity.

Determinations of disability are, in general, made by a state vocational rehabilitation agency or other appropriate state agency under an agreement between the Secretary of Health, Education, and Welfare and each state. This pattern of federal-state operation means that skills and the established organization of the state agencies and the existing relationship between those agencies and the medical profession will be utilized.

All of you know that disability evaluation is a highly complex process. The problem of administration is not as great when the criteria are to be applied by a compact group of evaluators, perhaps centralized in one spot or under uniform administrative direction. The problem becomes more difficult when decisions are made by widely dispersed teams under varying jurisdictions.

In evaluating disability on a mass scale, it has been essential to develop and to keep refining a method that will help to get the job done with greater facility and uniformity. For this purpose, we prepared (with the constructive assistance of the Medical Advisory Committee) evaluation guides, which contain clinical descriptions of over 130 disabling conditions that demonstrate great severity from the point of view of anatomical damage, functional loss, or residuals. In the absence of actual work or other evidence indicating an ability to work, the levels of severity that the guides describe for various conditions can be considered to constitute disability consistent with the statutory definition.

However, I cannot emphasize too strongly that the guides are just that, and we do not adjudicate with rigid or mechanical adherence to the clinical descriptions. The guides do not constitute a rating schedule. Each case is evaluated on its own merits. The decision whether an individual case comes close to an appropriate level of severity is, in all instances, one that requires the professional judgment of the evaluation team. Every state team includes in its membership a medical consultant, often a practicing physician who serves part-time as consultant to the state agency.

The determination of disability, while tightly anchored to medical determinability, is not exclusively a medical finding. The mere fact that an individual's disability falls somewhat short of the evaluation guides does not mean that he is *out*. As a practical matter, it is not possible to divorce *disability* from the vocational and personal context in which it is found. There is a group of cases which, when viewed from a medical stand-

point, do not permit a presumption that the individual is either clearly in or clearly out. In this so-called *grey area*, special attention is given to factors such as the individual's previous education and skills, his age, and his job adaptability. With the assistance of the state vocational rehabilitation agencies, we are attempting, on a case-by-case basis, to draw conclusions that may permit establishing a second set of guidelines (paralleling the medical guides) that will be applicable to nonmedical factors.

PROCUREMENT OF MEDICAL EVIDENCE

It is the applicant's responsibility to furnish basic medical evidence concerning his disability. The medical evidence needed to establish the nature and severity of his disability, the date it began, and its prognosis comes from the physician who has treated him and who knows his case, or the hospital or institution in which the applicant has been confined. Those of you who have already completed the medical report form for the disability "freeze" program know that it is designed to minimize the reporting burden on you. You are not asked to certify that your patient is or is not permanently or totally disabled. All you are asked to report is a brief history of your patient's impairment and to relate the clinical facts as you found them during treatment or examination of your patient. The medical report form is returned by the physician or institution to the social security office or to the state agency to avoid disclosure to the applicant or patient and protect the doctor-patient relationship.

As physicians, you can assist the disability program and your patient by furnishing the necessary medical reports. The evidence you furnish should be sufficient to enable a reviewing physician to make an independent diagnosis on the basis of clinical evidence supplied on the medical report. The type of medical report used is the same for the child's and for disability insurance payment purposes as for the disability "freeze." In neither case, however, is the use of the form mandatory, for the physician is free to use

the method of his choice. However, the items of information listed on the form furnish the physician with a guide as to the kinds of evidence needed by the state agency to make a determination of disability. By supplying the types of clinical evidence listed on the form, the reporting physician will reduce the need for additional reports that are sometimes requested when the evidence is not complete.

The medical report, based upon an existing medical record or upon a current examination, will, if it is complete and factual, ordinarily be sufficient to establish the degree of severity of the applicant's disability. In some cases there will be a need for additional medical information and occasionally an additional examination. The state agency that is responsible for making the determination of disability may need additional reports of diagnoses and clinical findings from existing records and will advise the applicant that he must secure these.

Where the initial medical report submitted by an applicant's physician fails to establish the severity of the impairment, the physician in the state agency may write directly to the applicant's physician for additional information. This approach has been adopted to preserve and strengthen the doctor-patient relationship. If the necessary information cannot be provided by the attending physician without a further examination, the applicant will be informed and asked to arrange for such an examination. Since the law places upon the applicant the responsibility for furnishing supporting evidence, he is responsible for paying any basic costs involved. However, a medical examination at the expense of the government may be authorized where, in the judgment of the reviewing physician in the state agency, it seems necessary to obtain additional evidence in order to insure that an improper award will not be made.

TIE-IN WITH REHABILITATION

While the primary aim in the disability field of the Bureau of Old-Age and Survivors

Insurance is to assure a proper determination of disability so as to provide protection against loss of income or loss of benefit rights, nonetheless, the disability evaluation process is intimately concerned with positive steps to restore impaired individuals to self-help and economic self-reliance.

One of the most significant features in the new social security law is the requirement that all applicants, whether for benefits or the "freeze," be referred promptly to their state vocational rehabilitation services. If sufficient appropriations are made available to these agencies, they will be in a position to provide many disabled persons with appropriate medical and vocational services that may prevent more serious disability or restore working capacity.

one should consider the criteria used by the program in evaluating severity of cardiac impairments and the degree such diseases disable the individual for any kind of substantial gainful activity, and the fact that the existence of a specific impairment is sometimes not well established in a denial, e.g., it may not be pertinent in a denial based on lack of work attachment.

The medical standards of the disability insurance program establish a high degree of severity for cardiac impairments. A level of residual severity while under therapy as defined by the American Heart Association's Class III C approximates the degree of impairment required. Established congestive heart failure in spite of therapy clearly meets the test of severity for cardiac impairments.

TABLE 57-1 NUMBER AND PERCENTAGE ALLOWED OR DENIED OF CARDIAC AS COMPARED WITH ALL OTHER IMPAIRMENTS, JULY, 1955-AUGUST, 1956

Type of impairment	Number			Per cent		
	Total	Allowed	Denied	Total	Allowed	Denied
All impairments	324,477	172,158	152,319	100	53.1	46.9
Cardiac impairments*	68,985	43,425	25,560	100	62.9	37.1
Noncardiac impairments	255,492	128,733	126,759	100	50.4	49.6

* Includes diagnosis under Code No. 401-447 of the *Manual of International Statistical Classification of Disease, Injuries and Cause of Death*, sixth revision, 1948.

Aside from the social desirability of restoring individuals to self-reliance, it is obviously to the advantage of the disability insurance trust fund that, if at all possible, the disabled person be restored to productive work. In line with these objectives, the law provides that an individual's benefit payments shall be suspended if he refuses without good cause to accept available rehabilitation services under a state plan. On the other hand, if he does undertake rehabilitation, he may continue to receive benefits for a *grace* period of one year, even though working, if his work is pursuant to an approved rehabilitation plan.

The tables show specific results on workers with selected cardiac impairments allowed and denied a period of disability July, 1955—August, 1956. In interpreting the tables

under the program. Severe and frequent angina in spite of treatment, coupled with electrocardiographic or clinical evidence of organic heart disease, is sufficient. When the impairment is solely of cardiac origin, the level of severity for an allowance is close to the clinical picture of established heart failure or equivalent.

Table 57-1 shows that the percentage of allowances for the cardiac impairment group is higher than the percentage of allowances for noncardiac or all impairment groups. The percentages for the different groups are:

- a Cardiac impairment group 62.9 per cent,
- b Noncardiac impairment group 50.4 per cent, and
- c All types of impairments 53.1 per cent.

These figures probably reflect the differences in the age distribution of persons with cardiac impairments as compared with persons with other types of impairments.

This is demonstrated by Table 57-2, showing the percentage of individuals in the

age of individuals in the younger age brackets of the denied group. The exact significance of this fact is difficult to evaluate because it can be influenced by a number of factors, such as the tendency to less severe heart disease in younger individuals, if

TABLE 57-2 NUMBER AND PERCENTAGE ALLOWED OR DENIED OF CIRCULATORY AS COMPARED WITH ALL OTHER IMPAIRMENTS BY AGE, JULY, 1955-AUGUST, 1956

Type of impairment	Number				Per cent			
	Total	Under 50 yrs	50-64 yrs	65+ yrs	Total	Under 50 yrs	50-64 yrs	65+ yrs
ALLOWED								
All impairments	172,158	33,838	94,595	43,725	100.0	19.7	54.9	25.4
Circulatory impairments*	47,094	2,137	28,907	16,050	100.0	4.5	61.4	34.1
Noncirculatory impairments	125,064	31,701	65,688	27,675	100.0	25.4	52.5	22.1
DENIED								
All impairments	152,319	24,759	88,820	38,740	100.0	16.3	58.3	25.4
Circulatory impairments*	30,372	1,943	19,306	9,123	100.0	6.4	63.6	30.0
Noncirculatory impairments	121,947	22,816	69,514	29,617	100.0	18.7	57.0	24.3

* Includes diagnosis under Code No. 401-468 of the *Manual of International Statistical Classification of Diseases, Injuries and Cause of Death*, sixth revision, 1948.

TABLE 57-3 NUMBER AND PERCENTAGE ALLOWED OR DENIED OF CIRCULATORY AS COMPARED WITH ALL OTHER IMPAIRMENTS BY SEX, JULY, 1955-AUGUST, 1956

Type of impairment	Number			Per cent		
	Total	Male	Female	Total	Male	Female
ALLOWED						
All impairments	172,158	147,624	24,534	100.0	85.7	14.3
Circulatory impairments*	47,094	40,043	7,051	100.0	85.0	15.0
Noncirculatory impairments	125,064	107,581	17,483	100.0	86.0	14.0
DENIED						
All impairments	152,319	120,271	32,048	100.0	79.0	21.0
Circulatory impairments*	30,372	23,974	6,398	100.0	78.9	21.1
Noncirculatory impairments	121,947	96,297	25,650	100.0	79.0	21.0

* Includes diagnosis under Code No. 401-468 of the *Manual of International Statistical Classification of Diseases, Injuries and Cause of Death*, sixth revision, 1948.

older age groups is higher in the cardiac impairment group as compared to the other groups. This fact holds true for denied as well as allowed cases. Such a result is consistent with the fact that circulatory diseases are more common in older individuals.

A comparison of the age distribution of denied and allowed groups with circulatory impairments shows a slightly higher percent-

effect of biological aging on the cardiac impairment group. The exact significance of this fact is difficult to evaluate because it can be influenced by a number of factors, such as the tendency to less severe heart disease in younger individuals, if the number of technical or nonmedical denials.

Table 57-2 records the number of allowed and denied individuals with circulatory impairments. The per cent of allowances by age group is 52.4 per cent for the under 50 years

age group, 60.0 per cent for the 50-64 years age group, and 63.8 per cent for the 65 and over age group. The fact that the percentage of allowances increases in the older age groups may indicate that aging factors increase the severity of impairment in cardiac cases or that the factor of age superimposed on a severe cardiac impairment tends to shift the determination in favor of an allowance of disability.

Table 57-3 shows that the percentage of females is higher in the denied group (21.0 per cent) as compared with the allowed group (14.3 per cent) and this fact prevails

The number of denied and allowed cases by sex are included in Table 57-3. The percentages allowed by sex are 62.6 per cent male and 52.4 per cent female. This result substantiates the difference described in the above paragraph.

Table 57-4 shows a preponderance (63.8 per cent) of cardiac impairments were of the arteriosclerotic and degenerative heart disease group (Code 420-422 of the Manual of International Classification of Diseases, Injuries and Causes of Death, sixth revision 1948). The next most frequent diagnostic group is hypertensive heart disease (Code 440-447),

TABLE 57-4 NUMBER AND PERCENTAGE OF CARDIAC IMPAIRMENTS ALLOWED AND DENIED BY MAJOR CATEGORY OF HEART DISEASE, JULY, 1955-AUGUST, 1956

Code*	Diagnosis	Number			Per cent		Distribution
		Total	Allowed	Denied	Allowed	Denied	
	Total number and percentage	68,985	43,425	25,560	62.9	37.1	100.0
401	Rheumatic fever with heart involvement	14	4	10	28.6	71.4	†
402	Chorea	14	4	10	28.6	71.4	†
410-416	Chronic rheumatic heart disease	4,029	3,215	814	79.8	20.2	5.8
420-422	Arteriosclerotic and degenerative heart disease	43,979	28,240	15,739	64.2	35.8	63.8
430-434	Other heart disease	2,306	1,437	869	62.3	37.7	3.4
440-447	Hypertensive heart disease	18,643	10,525	8,118	56.5	43.5	27.0

* Includes diagnosis under Code No. 401-447 of the *Manual of International Classification of Diseases, Injuries and Cause of Death*, sixth revision, 1948.

† Less than 0.1 per cent

with regard to circulatory impairments as well as noncirculatory impairments. This difference probably occurs because the technical or nonmedical denials are higher for females than for males because of the program requirements of recent labor force attachment.

Further, evaluation of the above result should include such factors as age distribution, severity of cardiac impairment and its effect on work adaptability, and number of technical or nonmedical denials by sex groups. In the program, the same medical standards are applied for both sexes. The medical criteria of severity of impairment for the cardiac diseases were selected to measure demonstrable cardiac reserve and are equally applicable to both sexes.

which includes 27.0 per cent of all cardiac impairments. The percentage of impairments meeting the required level of severity was higher (64.2 per cent) in the arteriosclerotic and degenerative heart disease group as compared with the hypertensive group (56.5 per cent).

The lower percentage of allowances for the hypertensive heart disease group as compared to the arteriosclerotic and degenerative heart disease group may be partially explained by the fact that the former group includes cases of benign hypertension, which generally do not meet the test of severity under the medical standards. Differences in age distribution and percentage of technical denials in the groups might account for this result.

TABLE 57-5 NUMBER AND PERCENTAGE OF CARDIAC IMPAIRMENTS ALLOWED A PERIOD OF DISABILITY BY MAJOR DIAGNOSTIC CATEGORY AND RACE, JULY, 1955-AUGUST, 1956

Code*	Diagnosis	Total No	Number			Total	Per cent		
			White	Colored	Other		White	Colored	Other
401	Rheumatic fever with heart involvement	4	3	1	†	100	75.0	25.0	†
402	Chorea	4	4	†	†	100	100.0	†	†
410-416	Chronic rheumatic heart disease	3,215	3,044	163	8	100	94.7	5.1	0.2
420-422	Arteriosclerotic and degenerative heart disease	28,240	26,963	1,215	62	100	95.5	4.3	0.2
430-434	Other heart disease	1,437	1,312	120	5	100	91.3	8.4	0.3
440-447	Hypertensive heart disease	10,525	8,647	1,833	45	100	82.2	17.4	0.4
	Total, cardiac disease	43,425	39,973	3,332	120	100	92.0	7.7	0.3
	Total, noncardiac disease	128,736	117,072	10,907	757	100	90.9	8.5	0.6
	Total, all impairments	172,161	157,045	14,239	877	100	91.2	8.3	0.5

* Includes diagnosis under Code No. 401-447 of the *Manual of International Classification of Diseases, Injuries and Cause of Death*, sixth revision, 1948.

† No cases in sample.

According to Table 57-5, the percentage distribution by race and type of cardiac impairment of individuals showed a fairly uniform rate except for the hypertensive heart disease group. In the latter group, the percentage of colored is substantially higher (17.4 per cent) as compared with a percentage range of between 4.3 and 8.4 per cent in other diagnostic groups, and 8.3 per cent for all impaired individuals included in this study. This result is consistent with re-

ports in medical literature that the incidence of hypertensive heart disease is higher in colored as compared with white groups of individuals.

Perhaps the data presented will be of some value to other disability insurance programs and to this group which is concerned, as the disability programs are, with the very difficult task of evaluating the cardiac patient's ability to work.

REPORT OF PANEL ON WORKMEN'S COMPENSATION

Rodney R. Beard

The first concern of the meeting and of this panel was the rehabilitation of the cardiac patient. Workmen's compensation laws and practices have both favorable and unfavorable aspects for rehabilitation. For many cases they provide the means by which rehabilitation is supported, especially in the medical phases, and they may offer the framework to stimulate and administer comprehensive rehabilitation programs. On the negative side, rehabilitation is sometimes hindered by the delay associated with long periods of litigation, during which the claimant is motivated to maintain his disability. A second concern of this panel was equity in workmen's compensation cases involving heart disease. We sought information as to the quickest and best ways of arriving at fair awards for such claims. A third interest was the evaluation of disability in heart disease under disability insurance programs.

Dr. Harry Ungerleider reported that heart

disease is highly prevalent, some ten million Americans having some recognizable form of the disease, and fully half of the middle-aged productive workers are affected. There are over a million persons who suffer attacks of coronary occlusion each year, of whom more than half return to the labor force. Among workers with no medical complaints or apparent symptoms, evidence of cardiovascular disease has been found in 15 per cent and in half of these there were clinically significant disorders. Coronary and hypertensive diseases accounted for over 10 per cent of these.

There are many evidences that persons with heart disease perform well in industrial employment, not being significantly better or worse than others with respect to absenteeism, productivity, and accidents. However, there is evidence of widespread reluctance to employ persons who have had heart attacks or other manifestations of heart disease. A major factor is said to be the threat of monetary loss arising from workmen's compensa-

tion claims. There was extended discussion of second injury laws as a way to encourage the employment of persons with heart disease. New York has a law that has been shown to be effective, but it is not widely known to employers. Further experimentation in other states and wider knowledge of the protection offered to employers by such laws appear to be indicated.

In further discussion subsequent to the presentation of Dr Ungerleider's report on the magnitude of the problem of the cardiac in industry, it was stated that over half of the middle-aged productive population has heart disease and that no adequate study of the extent and consequences of this in any given industry has been made. Evidence that interest in this field is not new was given, in particular the fact that a night clinic for cardiac workers was established in New York City as early as 1911 by Dr. Hubert V. Guile. It was from this clinic that the New York Heart Association developed, and gave the leadership to start the American Heart Association.

Dr Rodney R. Beard raised a question as to the true importance of the workmen's compensation risk as cause for rejection for employment of cardiac patients. It was suggested that in many instances age or other factors are the true reasons for rejection, but that the presence of heart disease is given as the excuse. Dr. Beard raised the question of the importance of the attitude of rejection that is present as an unconscious feeling on the part of many individuals toward crippled or otherwise handicapped persons. Dr. Ungerleider brought out the importance of keeping the diagnosis of heart disease or other disease confidential. It should be given only to the top management and not be permitted to become widespread knowledge among the patient's fellow workers. He also brought out the importance of avoiding unnecessary mention to patients of insignificant murmurs, premature systoles, dizziness, or high blood pressure because of the unnecessary anxiety produced by their knowledge of such factors.

Mr. Barnett S. Fox asked Dr. Ungerleider if it be true that persons with heart disease

"can work, do work, and should work," and should there be any concern about continuing medical supervision? His inference was that such persons must be under some danger of a heart attack, otherwise the continuing medical supervision would not be necessary, nor should they be restricted as to their work. Dr. Ungerleider agreed that there was a danger of heart attack in such persons and that it was important that they be placed in work situations that were within their capacities, and with continuing medical guidance to be sure that they stayed at work within their capacities. There is also the point that they should be so placed in work that they would not endanger others, if fainting or other incapacity occurred while they were operating machinery or vehicles.

Mr. Henry Sayer gave a resume of the developmental history of workmen's compensation laws, which embody the unusual principle of responsibility for the effects of injury without fault for the occurrence. Better understanding of the background and philosophy of these laws will help to clarify the role of the physician in his work with claimants and in industrial commission proceedings. There are forty to fifty million American workmen covered by such laws. They receive benefits for disabilities *arising out of and in the course of employment*. Aggravation of a pre-existent defect is generally accepted as an injury for which compensation will be paid, if something in the man's work caused the aggravation. His report was received with general approbation for its judicious selection of essential information. A correction was offered, it being said that the number of workers covered was estimated by the Social Security Administration in 1954 to be thirty-nine million, not the higher figures he quoted.

Dr. Meyer Texon reported on causal relationships. The discussion centered on coronary artery disease. He showed how considerations of fluid mechanics can explain the localization of atherosclerotic lesions. The composite effect of velocity of blood flow, external attachments of a vessel, caliber of the lumen, the degree of curvature, and the

branching characteristics determine sites of predilection. In the subsequent discussion, Dr. Louis E. Viko asked if there was a relation between the occurrence of coronary occlusion and the suddenness of exertion. To this, Dr. Texon answered, "In exceptional cases, yes." He went on to say that if one can get a well-documented history, there will invariably be a story of premonitory symptoms before a heart attack. Dr. Viko felt that although this may be common, it was not invariably the case. Dr. Texon agreed to this. Mr. Fox pointed out that the thresholds of pain are different, in that the discomfort that may be reported as a symptom by one individual may be totally disregarded by another. Dr. Ungerleider raised questions about the influence of the heart rate upon the rate of blood flow and upon the localization of atheromatous plaques. He pointed out that in bradycardia there is definitely a slowing of blood flow, yet it is associated with coronary accidents. He made the point that many coronary accidents occur during sleep when the blood flow is at the lowest rate. Dr. Texon answered that there were several variables, the size of the lumen of the artery being of primary importance. There is no strict correlation between heart rate and velocity of blood flow. He said that congenitally wide arteries may occur, in which case the vessel can carry a large volume without a rapid flow rate. He also said that we all have different fabrics, that some people develop greater fibrosis than others in scars, but that notwithstanding these matters, these points of predilection still apply.

Sequences of events that would establish a causal relationship between occupation and a heart attack in a person with pre-existing pathology were discussed at length. All agreed that if symptoms appeared during exertion, or within two hours, that a causal relationship must be assumed. Some members of the panel would accept attacks occurring within twenty-four hours of exertion and others felt that there might even be a period of several days. In a later discussion with some members of the panel on pathology, it was asserted that this kind of distinction re-

sembled arguments about the number of angels who can dance on the point of a pin and that it is unfair to deny assistance to the man whose attack begins while he is away from work, while compensating the one who happens to be on the job when it occurs. There was extended discussion of the role of exertion in precipitating a heart attack and many questions were raised as to whether unusual exertion was necessary. This got into a semantic argument and it appears there could be no agreement on what constituted an unusual exertion. The conclusion was that exertion can precipitate a heart attack. It was pointed out that, to be considered by a referee, there would have to be an "accident" within the meaning of workmen's compensation law, and that such would not exist in ordinary accustomed activities. When it is established that an accident has occurred, this being the decision of the referee, the physician must accept this as an accident and give his opinion on causal relationship.

Dr. Irvin Klein discussed the work placement of cardiacs. He reminded us that very few jobs call for the use of full cardiac capacity and that there is usually sufficient reserve to satisfy work demands after a heart attack. He emphasized the importance of returning the patient to a paying job as the goal of rehabilitation. Education of physicians, government, management, and labor is necessary.

Mr. Kenneth Pohlmann described workmen's compensation as "an ailing form of social insurance," which despite earnest attempts to bring it into focus with the changing social and industrial order has remained "bogged down in a morass of piddling reforms." Workmen might be better off to resort to suits at common law, since, in general, workmen's compensation serves to limit the employer's liability more than to help the worker. Public interest in comprehensive rehabilitation, as reflected in Congress, is at a high level. Organized labor has made rapid gains in benefits and health insurance for nonoccupational diseases. It is not inconceivable that this expanding field will, in time, take over the need. It is time to con-

tion claims. There was extended discussion of second injury laws as a way to encourage the employment of persons with heart disease. New York has a law that has been shown to be effective, but it is not widely known to employers. Further experimentation in other states and wider knowledge of the protection offered to employers by such laws appear to be indicated.

In further discussion subsequent to the presentation of Dr. Ungerleider's report on the magnitude of the problem of the cardiac in industry, it was stated that over half of the middle-aged productive population has heart disease and that no adequate study of the extent and consequences of this in any given industry has been made. Evidence that interest in this field is not new was given, in particular the fact that a night clinic for cardiac workers was established in New York City as early as 1911 by Dr. Hubert V. Guile. It was from this clinic that the New York Heart Association developed, and gave the leadership to start the American Heart Association.

Dr. Rodney R. Beard raised a question as to the true importance of the workmen's compensation risk as cause for rejection for employment of cardiac patients. It was suggested that in many instances age or other factors are the true reasons for rejection, but that the presence of heart disease is given as the excuse. Dr. Beard raised the question of the importance of the attitude of rejection that is present as an unconscious feeling on the part of many individuals toward crippled or otherwise handicapped persons. Dr. Ungerleider brought out the importance of keeping the diagnosis of heart disease or other disease confidential. It should be given only to the top management and not be permitted to become widespread knowledge among the patient's fellow workers. He also brought out the importance of avoiding unnecessary mention to patients of insignificant murmurs, premature systoles, dizziness, or high blood pressure because of the unnecessary anxiety produced by their knowledge of such factors.

Mr. Barnett S. Fox asked Dr. Ungerleider if it be true that persons with heart disease

"can work, do work, and should work," and should there be any concern about continuing medical supervision? His inference was that such persons must be under some danger of a heart attack, otherwise the continuing medical supervision would not be necessary nor should they be restricted as to their work. Dr. Ungerleider agreed that there was danger of heart attack in such persons and that it was important that they be placed in work situations that were within their capacities, and with continuing medical guidance to be sure that they stayed at work within their capacities. There is also the point that they should be so placed in work that they would not endanger others, fainting or other incapacity occurred while they were operating machinery or vehicles.

Mr. Henry Sayer gave a resume of the developmental history of workmen's compensation laws, which embody the unusual principle of responsibility for the effects of injury without fault for the occurrence. Better understanding of the background and philosophy of these laws will help to clarify the role of the physician in his work with claimants and in industrial commission proceedings. There are forty to fifty million Americans workmen covered by such laws. They receive benefits for disabilities arising out of and in the course of employment. Aggravation of a pre-existent defect is generally accepted as an injury for which compensation will be paid, if something in the man's work caused the aggravation. His report was received with general approbation for its judicious selection of essential information. A correction was offered, it being said that the number of workers covered was estimated by the Social Security Administration in 1954 to be thirty-nine million, not the higher figures he quoted.

Dr. Meyer Texon reported on causal relationships. The discussion centered on coronary artery disease. He showed how considerations of fluid mechanics can explain the localization of atherosclerotic lesions. The composite effect of velocity of blood flow, external attachments of a vessel, caliber of the lumen, the degree of curvature, and the

less civilized circumstances would lead to a fist fight. They must leave the loser satisfied that he has had a fair chance to win his point. This function of proceedings in law should not be forgotten nor discounted.

In subsequent discussion, Professor Rutledge was asked what could be done when all the expert opinions added up to "I don't know." To this he answered that it then became a matter of turning to the legal precedents to see where the burden of proof might be. Dr. Klem commented upon experience in New York, wherein a board of three consultants in the area of pulmonary disease was established many years ago. The members are chosen for competence and established prestige. They see almost every case of disease of the chest and supply technical medical opinion. They have available to them the results of field industrial hygiene surveys done by skilled workers of the Industrial Hygiene Bureau. Due process of law is maintained, the Board being subject to cross-examination. It is felt that the system has worked out well in New York State simply because it is a one-unit panel. If there were several different panels, it might not have been nearly so successful. There might have resulted different conclusions and different precedents among the different boards that would confuse the issue. It was emphasized that this is a board limited strictly to occupational pulmonary disease. It was also emphasized that the panel members are on the State payroll and are prohibited by law from treating compensation cases. The intention is to maintain the impartiality of the specialist's position, in that he does not represent another party in a litigation later. An early problem of the New York Compensation boards was in finding impartial experts. A rather scandalous situation had arisen in which experts began doing thriving businesses either with unions or with insurance carriers. When the 1932 law on pneumoconiosis developed, it was hard to find a fair, equitable way of handling it. Three fair impartial experts were appointed from the staffs of the medical schools. After these appointments, the law was amended so that no doctor so employed by a commission

for the examination of such cases could examine for any insurance carrier or practice in any other field of compensation cases. This was not an exclusion from the practice of medicine, but from giving opinion in compensation cases. These experts can only advise. Their findings are not conclusive nor mandatory upon the board. The experts limit themselves to a position where they must refuse to find how an accident happened or even that it did happen, and insist that those matters of controversy be decided before they arrive at a medical conclusion.

The question was asked, If the panel system had worked so well for pulmonary disease, why had its use not been extended to other fields? It was reported that an effort had been made to do this, but failed because there were not enough qualified experts who would accept the conditions required for the work. It was said that organized medicine and the insurance carriers had opposed the development of expert panels.

The point was made that the discussion was missing the main issue, the welfare of the injured person. There should be continuity of competent medical care from the beginning of illness to completion of rehabilitation, and separation of different functions as observed in the relationships under discussion was not consistent with this ideal.

Dr. Beard reported that in California the Industrial Accident Commission has a medical department, the head of which selects a panel of independent medical experts. The referee may ask for help from this panel and it is given with the consent of the consultants. The insurance carrier always pays the bill because the commission has no money for this. The opinion of the expert is not binding, but when requested is usually followed. Less than half of the referees in the San Francisco area use this medical examination.

Another way of getting unbiassed medical opinion is the use of the so-called "arbitration upon examiner." He is a physician selected by joint agreement of claimant and defendant, to make an independent examination, his selection being based upon his reputation for fairness. Some of the referees in the

this is an excellent way of arriving at proper medical testimony

In conclusion, it was felt that the New York plan has the virtues of three high-class clinicians with expert knowledge and the ability to concentrate all cases of pneumoconiosis and pulmonary diseases in these three physicians, thereby increasing their competence in this small segment of medical illness. It was pointed out that the subject of pneumoconiosis is complicated by the fact that disability is defined by law in 13 states and a medical panel would have to have considerable knowledge in those states as to the legal definitions involved in this diagnosis. Furthermore, the silicosis laws are obsolete and are based almost solely upon x-ray studies. We know from more recent cardiopulmonary function studies that actual functional correlations vary to a greater or lesser degree from the x-ray films. Basically, then, the laws dealing with silicosis are in need of revision. In dealing with heart disease, it is most important that we avoid the arbitrary errors that occurred in the development of laws for handling silicosis.

Mr. R. G. Knutson, Chairman of the Industrial Commission of Wisconsin, said that although physicians are entirely honest, they find themselves being associated generally with claimants or defendants in a series of cases. Impartiality is hard to maintain and it becomes difficult to place proper value on medical evidence. It would be of great assistance to administrators if a conference such as this could set up some guideposts for administrators. Thoughtfully developed rating schedules for consideration of various disabilities have proved valuable, even though they have had to be arbitrary in some respects. This should be done for heart disease. Flexibility in administration and ability to change or abandon established formulas is essential. It was agreed that the eight questions listed by Mr. Knutson in his prepared paper did indeed state most of the problems that need to be resolved for more effective administration of workmen's compensation laws in the existing framework of medical and social relationships.

There was a long discussion of the use of panels and the selection of impartial experts, the Utah experiment being the type example, presented by Commissioner Otto Wiesley and Dr. Louis Viko. The essentials of this plan rest in the way it was conceived and developed. A workmen's compensation commissioner, distressed by the inequities of awards made under an adversary system of medical testimony, discussed the problem with leaders in medicine, labor, and management. They agreed upon a system by which medical facts and opinions are reviewed by qualified experts working together and reporting their findings in their own way. The essential benefits of the adversary system are maintained by making the experts available for cross-examination. The result has been a notable reduction in strife and avoidance of delay. All agreed that this is an admirable system in Utah, but there were some doubts that it could readily be developed in other places, for the key to its success is the interpersonal relationships of certain individuals. When such leaders can be found in other states, similar or different systems may be developed with similar success. It was suggested that similar results could be had if physicians insisted that they be permitted to make uninhibited, thorough examinations and be allowed to report all their findings to the Commissioner without interruption or interference by attorneys for either side. The members of the panel who represented the legal profession stated that this could not be accomplished. Those present from Eastern states expressed the opinion that a system such as that in Utah would not be acceptable in the more highly industrialized areas of the country.

Every member of this panel has expressed genuine enthusiasm and deep appreciation for this meeting. One does not often have the opportunity to talk in comfortable, informal circumstances in such distinguished company as has been assembled here.

MR. BARNETT S. FOX: I would just like to amend that portion of Dr. Beard's report which calls for the education of the physician, government administrator, labor and

management definitely to include the education of the lawyer.

DR. LEWIS H. BRONSTEIN: One point was brought up in the Workmen's Compensation Panel, I think, by Dr. Price, that in those industries where there is a comprehensive security program the incidence of workmen's compensation claims is very low. I believe this supports some of the statements that were made in the summary report that in many cases the claimant comes to a compensation claim as a matter of revenge

against the employer because of an injustice which he feels has been committed in his particular case

DR. LEO PRICE: The only comment I can make is to emphasize what has been mentioned by Dr. Beard. With a very large population of rather litigious people, I do not believe we have had enough compensation cases in forty years to count on one hand. The primary reason for this is that there is job security. The employer has to take them back

PART VI OUTCOMES AND PROSPECTS

CONFERENCE SUMMARY

Maurice Visscher

My function clearly resembles that of the elastic reservoir. I am to take up whatever time is left and not occupy any more. To the physiologist the more conventional expression for the elastic reservoir when treated in connection with the vascular system is *wind kessel*. However, I trust that I will not go into the wind chamber act in this connection.

I believe it is by now clear to everyone who has participated in this Conference that the present state of our knowledge about work and the heart is very unsatisfactory in many respects. I do not propose to make a review of the preceding reviews during this next half hour because I believe that would be pointless. It occurs to me that when one begins redistilling knowledge too often, a fair share of it gets lost in the vapor. However, it is undoubtedly worth our while to take stock of our current situation and to see in broad terms what this conference has performed and what we should like to see done in the future. In our small groups we have dealt with the evaluation of information in fields in which each of us has had some fairly

long experience and, particularly in this session today, though each of us has his separate horizon, we have had an opportunity, through the bringing together of individuals in a variety of disciplines, to broaden our horizons a great deal. I think it has been quite an interesting thing to bring together fifty or sixty experts in as widely different fields as have been brought together here for this Conference and to let them hear something about each other's specific problems.

Perhaps the most important element in our activities has been to identify our areas of ignorance as well as our areas of information, because it is only when we recognize our specific problems of ignorance and define those problems that we as scientists are able to go at a solution of them in any intelligent way. A clearer definition is the first step in the solution of the problems. I do not believe that anyone dreamed that this Conference was going to come out with any encyclopedic recapitulation of facts about work and the heart. This was not the order that was really written when this Conference

was planned and it would have been entirely hopeless to do such a thing. However, we can ask ourselves whether we are happy with the more modest results that have come out in connection with the integration of such information as we have been able to discuss together and, as I pointed out a moment ago, the identification of the problems that are before us at several levels on scientific scores and on the social score. This Conference differs from any that I myself have attended before. I have attended many conferences in which basic scientists have co-operated in the discussion of problems with persons interested in the application of science. But as applied scientists, today we have put a third dimension into our considerations, the dimension of the consequences of our problem to the community as well as the social aspects of the problems of the diseases of the heart. I have tried to analyze this program in a couple of different ways. Since we scientists who are meeting with nonscientists interested in the same problems are unfamiliar with thinking about these problems from a social viewpoint, I sat down and tried to put our problems into a social frame. It seems to me that from a social viewpoint our problems in connection with work and the heart are really of three types. First, those that are related to prevention of diseases of the heart. Second, those concerned with the salvage of the patient or person who happens to have disease of the heart. Third, protection of the worker who is dependent upon society in the face of disease of the heart.

When we have said this we have only set down certain categories into which we are going to have to put the various types of problems that we have before us. Therefore, I want to turn from this classification to another with which it may be a little simpler for us to deal as we consider how we should move as we take our next steps. If you like, this may be said to be in the various levels of scientific complexity. As you heard from the report of the recorder for the panel on basic physiology, some of our problems deal with fundamental chemistry, physics, and

anatomy. They go to the bottom of the most basic things with which we have to deal in connection with living organisms. Obviously, we are not going to solve the problems that arise from the social analysis, that is, the prevention of disease and the salvage of the patient with disease, without learning more about the fundamental mechanisms by which the heart muscle acts and the fundamental mechanisms that are involved in the growth, development, and survival with age of the vascular bed. To a very large degree, these are chemical and physical problems. As you heard from Dr. Stewart's report, the experts here who made up the panel on basic physiology have to report to you that, unfortunately, we do not know, at the present time, exactly what the chemical reactions are that go on in a piece of heart muscle when it contracts and when it recovers from contraction. We know a great deal about some of the aspects of the chemistry and energetics of heart muscle but, even today, it is uncertain whether it is at the expense of adenosine triphosphate, the ATP about which you heard, that contraction actually occurs. We do not know what all the possible sources of energy for producing shortening of heart muscle may be. There are numerous other high-energy phosphate bond compounds in muscle about which we know, and there are probably others about which we do not know at the present time. Obviously, it would be impossible to do much in the study of the effects of various things in which the applied scientist is interested concerning heart muscle until we do learn more precisely what the fundamental chemical and physical mechanisms are in that structure. To take an example, today it is not possible to say precisely at what point digitalis glycosides enter into the chemical reactions in heart muscle to produce improvement in the work capacity of the heart. This may be something of a shock to those of you who are outside the field of more fundamental research in muscle and heart muscle chemistry, but it is a fact and I think it would be well for us, as well as for the expert witness on the witness stand, to learn to say a little more frequently

and a little bit more graciously, "I do not know." The recognition of ignorance is very important from many points of view.

When we go from this more fundamental stage to the stage of more integrated physiology and pathology, we find numerous other problems, to some of which I shall refer in a moment. Then if we go to the next step on the clinical side, we find ourselves in just as much difficulty. I believe the controversies indicated this morning about the usefulness of various measurements of one or another parameter of cardiovascular and muscular functional capacity in intact men are perfect testimony, if you like, to the fact that we have too little information to be able to make the type of critical and specific evaluations that we would like to make in order that with such quantitative data we can give more information than the trained physician can gather, as he works as an observer using rough and crude methods in the evaluation of disabilities and work capacity and in the prognosis of the patient and worker with heart disease. We are at the beginning of a long program of additional work in connection with the subject of our conference. It would be very naïve for us to believe that the information currently available is going to do us a great deal of good in actually solving these many problems which have been brought up here. It would be equally wrong to be pessimistic about the possibility of making great progress in these areas, if the approach is made from the more fundamental, scientific point of view. Now in the various discussions of the morning, a number of unsolved problems have been mentioned and in the particular panel with which I was associated we spent a good deal of time yesterday discussing and pointing up important unsolved problems. One that was mentioned by practically everyone on the panel on basic physiology is one to which I have already referred, namely the absolute necessity for encouraging work on the fundamental biochemistry of heart muscle. Until we understand more in detail the enzymatic reactions, the sources of energy, the fuels, and other related matters about the more intimate

nature of muscle contraction in the heart, we fundamental scientists are not going to be able to provide to those working in applied science and the laymen interested in these problems the answers that they must have before many of the practical questions can be solved.

Many specific problems were mentioned yesterday, and I want to repeat a few of them. The questions included such as these. Would it be possible to develop better cardiac function tests in man which would distinguish sharply between heart muscle function and other limiting capacities in man? Would it be possible to develop methods for the detection of atheromatous plaques in living organisms without the necessity of microscopic observation? Are there other ways in which one could detect those individuals who constitute, as Dr. Edwards said, a large fraction of the older patients who come to autopsy and who have severe coronary atherosclerosis? It will be necessary to determine, in connection with cardiac function tests, the role of nonexercise stress factors. It will be necessary to ascertain, in connection with the causation of the accidents of coronary occlusion, whether other stress factors such as emotion do play a role. These are just a few of the many suggestions for specific research projects that have been discussed and I shall not take the time to go into more of them at this moment.

In a very broad way what are the outcomes of this Conference? In the first place, it seems to me that there has been a broadening of the perspectives of a large group of people. As I indicated a little while ago, this type of conference does bring about a cross-fertilization across disciplinary lines and one would hope that it will help in setting the stage for public interpretation of the nature of our problems in relation to work and the heart at the basic scientific, the clinical, and the social levels. I believe it is quite clear that out of this Conference comes the recognition that more education of the public is going to be an important factor in helping to salvage patients with heart disease and helping in the protection of workers with

heart disease who are dependent on society at large. I am sure that education is a function that is not going to be carried out largely by ourselves. It must involve the co-operation of other interested agencies. I was interested in the suggestion that was made this morning by one of the panel chairmen that the American Heart Association might be encouraged to do even more than it has along the lines of education in this regard. I feel very much incapable of dealing with any of the broader aspects of the ideas concerning social legislation that were brought out this morning. Nevertheless, I feel that it is encouraging that out of this Conference there have been developed broader views as to how we in our American society, and perhaps world society, can do something at the level of social legislation to bring about improvements in the situation with which we are dealing. Dr. Keys referred to a specific suggestion made by the clinical physiology group. It was suggested that an international study committee be established which might operate over a period of time to try to get some clearer definitions and greater uniformity in the study of methods of assay of fitness. It seems to me that this would be a worthwhile accomplishment, if it could grow out of the activities of the Conference. The suggestion that in our medical colleges we could introduce more significant programs of study in relation to work classification units in relation to disability from heart disease and other disease also seems to me to have great merit.

Finally, I think the possibility of a Second Conference on Work and the Heart some day in the future seems altogether desirable. This would be particularly true if some time were to elapse, so that some of the seeds sown here in the last two days may germinate and grow. I would hope that those seeds could grow in many directions.

We have spent two and a half days together, and I am sure each one of the participants in this program would like me to speak in his behalf in appreciation of what the organizers of this Conference and its sponsors have done in making it possible. I

personally have heard nothing but praise for the general plan of the Conference, although, at first glance, the heterogeneity of the subject matter from physical chemistry to sociology might seem to be a barrier to useful results. In reality the Conference did allow us opportunity for detailed treatment of specific subjects and the small size of the panels that we have had allowed their members to get to the heart of many problems in informal discussions. I have been impressed by remarks that several of the conferees have made as to how satisfying it was that there was so little talk on generalities and so much talk that was to the point in the small panels. I think that this Conference has been worthwhile on two scores. Scientists have been brought face to face with their colleagues in their own specific problem. They have also been brought face to face with the sociologic consequences of heart disease. In this I am speaking mainly for the scientists and I hope that the people interested in the more social aspects of the problem feel equally rewarded in having been able to confer with experts in the more scientific fields. Finally, I believe I express the hope of all of us that this may not be the last Conference. Not that we would suggest that the same groups of people be brought together, but the subjects presented should be reviewed again by groups of experts in these same fields.

DR. BELKNAP: We are all very grateful for the work, co-operation, and really unselfish devotion that all of you have manifested in helping us make this Conference the success that Dr. Visscher has implied. It has been stimulating to hear your discussions over the table and other times outside the formal meetings.

We appreciate, particularly, your interest in this common social cause of which Dr. Visscher has spoken so well. I believe we will profit by going back to the quotation of Dr. Andrus at our opening dinner when he emphasized in his scripture quotation the word *Hope*. We are thrilled that some of you might wish to come with us on another tour beyond the horizons in a second conference.

EPILOGUE

FINAL GREETING TO THE PARTICIPANTS BY THE DIRECTOR OF THE FIRST WISCONSIN CONFERENCE ON WORK AND THE HEART

We appreciate the apparent satisfaction expressed by all the participants in the conduct of the Conference. I was particularly interested in the thread that ran through all of your discussions on the prevention of disease and the salvage of the cardiac patient. This certainly epitomizes the responsibility and the art of the physician.

We are all very grateful for the work, cooperation, and really unselfish devotion that all of you have manifested in helping us make this Conference a success. It is astounding that so many of you have spoken to me of kindred interests even in the representatives of so many disciplines who have been brought here. We feel that we have accomplished much in arranging the meeting of the minds of you scientists.

As we bring this First Wisconsin Conference on Work and the Heart to a close we can make these five observations:

These conference papers cover the well-

digested current opinion of the leaders in the basic and clinical research aspects of the relationship of work and the heart.

It is obvious that specific answers to many of the problems involved are not yet available.

We must look to the future for the development of further avenues of research suggested by this working conference.

It is our earnest hope that many of the participants in this conference will reassemble here in a few years to assess again in this keen and friendly type of debate the relationship of work and the heart.

It is for this reason that we have designated this the First Wisconsin Conference on Work and the Heart. I would echo Dr. Visscher's closing suggestion, "that the subjects presented here should be reviewed again by groups of experts in these same fields."

ELSTON L. BELKNAP, M. D.
Director of Conference

INDEX

- Absenteeism, and attitude of industry, 421, 422, 507
 Bureau of Labor Statistics, 419
 and compensation nervous, 470
 in Consolidated Edison study, 419
 Gillette Safety Razor Company, 385-386
 recurrence, 398-399
 in Refinery employees, 373-374
 and vocational guidance, 434
 work time lost, 344, 373
 Accidents, and cardiacs, 385, 386, 488, 491, 507
 509
 definition, 494
 prevention and Compensation system 441, 443
 and property damage, 421
 Utah Medical Panel System, 482, 498
 See also Injury
 Acclimatization, and heart rate, 192
 Acetylcholine, 175
 Actomyosin, and adenosine triphosphate 10, 11, 99
 Acute attacks, 331
 and awareness, 390-391
 danger, and medical supervision, 508
 and exertion, 296, 495, 508, 509
 medicolegal etiology, 299
 painless, 390-391
 and pre-existing defect, 428-431, 481, 492-493
 survival and return to work, 411
 Adaptation, 158-169
 to oxygen tensions, 208
 to work loads, and blood supply, 258-261
 and cardiac output, 208
 and heart rate, 181
 and muscle responses, 258-259
 and training, 158, 209, 226
 Adenosine triphosphate, and energy yield, 8-16, 518
 and mercury, 9-12, 16
 and solubility of actomyosin, 10-11, 99
 specificity of action, 13-14
 Adrenaline, 175
 Adrenocorticotrophic hormone, 77, 78, 80
 Age factor, 132-139
 and blood pressure, 127, 133-137
 calcification of aorta, 418
 in cardiac output, 82, 133, 134, 138, 139
 in cholesterol levels, 108
 and coronary occlusion, 283-287
 and disability 404, 481, 499, 501-505
 and electrocardiographic findings, 273, 384
 and employment, 106, 277-279, 297-298, 308,
 313-317, 318, 320, 369, 404-406, 409
 and exercise 133-137
 skung, 200-206
 heart disease, 313-317, 384, 399, 504-505
 arteriosclerotic, 119, 127, 234, 259, 285, 286,
 371, 373, 418, 507
 coronary, 104-106, 107, 259, 283-287, 289,
 418
 functional, 172-173, 174
 hypertensive, 354, 384
 Indiana farmers, 119-121
 productive population, 508
 rheumatic, 354, 384
 and heart rate, 71-72, 185
 of hemorrhages, and atherosclerosis, 217
 and myocardial infarction, 108, 270-271, 274
 276, 277-279, 315, 320, 376-378, 381, 381,
 388-390
 and collateral circulation, 389
 and survival, 392-393
 in neurocirculatory asthenia, 170, 172, 173, 174
 and off-job stresses, 294
 and oxygen consumption, 136
 arteriovenous difference, 82
 and peripheral resistance, 133, 135, 139
 and physical fitness, 177-179
 and rehabilitation, 351, 354, 357, 358, 363

Age factor (*Contd*)

- and rheumatic fever, 104
- and stroke index, 133, 134, 135, 137, 138
- and sudden death, 283-287, 297-298
- and thrombotic occlusion, 285-286
- and waist size, 106
- and work classification, 123, 308, 323-324, 331, 344

Alameda County Heart Association, 341

Alaska, Workmen's Compensation laws 441

367

371,

Anemia, 49, 51, 77, 78

Angina pectoris, 404

- in arteriosclerotic heart disease, 371
- in calcific aortic stenosis 372
- development 496
- and employability, 314, 318, 319-320, 345, 371, 372
- and time before work resumption, 374
- in hypertensive heart disease, 371
- improvement, with work 314

1, 389-390

503

- rehabilitation, 362, 363
- in rheumatic heart disease, 371
- and selective placement, 371
- and stress cold in cardiac farmer, 213
- physical 294

Anoxia, 51, 235, 239, 241

Antigravity suit, 55-56

Anxiety, and dyspnea on exertion 144

electrocardiographic complexes 164-165

and exercise, 162, 333

tests, 143, 144, 146

and heart, 146, 222

and employability, 335

and myocardial infarction, 388

in neurocirculatory asthenia 171

, 252,

and cardiac oxygen consumption, 21, 30-32, 87

tension-time index, 27, 29

coarctation, 251

and coronary blood flow, 51

endocarditis, 255

murmur, and work classification, 372

regurgitation, 99

and blood infusions, 40, 42

hemodynamic effects, 34-46

superimposed on mitral, 42-43

in rheumatic heart disease, 372

sclerosis, 251, 252

stenosis, 51, 372

syphilitic involvement, 123, 252

Arfonad, 82-83

Armed Forces Institute of Pathology, autopsy findings, 464

Arrhythmias, and circulatory stress, 294

fatal, and cardiac standstill, 340

in neurocirculatory asthenia, 171

and selective placement, 371, 372

and work classification, 333, 371, 372

and conduction system, 251-252

Arteritis, healed, and bundle branches, 252

Arthritis, 332, 363

Asthenia, neurocirculatory, emotion and exercise,

160-162, 164

and physical unfitness, 170-176

vasoregulatory, 171

Atherosclerosis, 259

and cardiac status after employment, 337

coronary arteries 118, 283-287, 290-292, 297,

300, 430, 431

etiology, 168, 233-241, 296-297, 300, 301, 380

cholesterol. *See* Cholesterol

encrustation theory 239-241

Winternitz theory, 301

and exercise tolerance test, 333

grading, 234-235

and hemodynamic changes, 429-431

localization of lesions, 508

and blood flow, 428, 509

and mortality, 118, 430

sudden death, 290-291, 292, 300-301

myocardial infarction, 290-292, 297

and occlusive disease, 283-287, 431

progressive, 300-301

and physical activity, 111, 224, 285, 300, 301,

334, 337

post-mortem findings, 290-291

and thrombosis, 236

1-429

See also Training

Atrioventricular node, 251, 252-253, 255

Atropine, 56

Ballistocardiography, 159-160, 162, 165, 171

Baton Rouge Refinery, 368-374

Beckman Hospital Unit, 227

- Bellevue Hospital, evening cardiac clinic, 120
 Work Classification Unit, 227, 305-310
- Benzodioxane, 167
- Beriberi, 77, 78, 79, 90
- Bernoulli's principle, 247-248
- Block, arteriovenous, 175, 333
- atrioventricular, 252, 255
- in cardiac factory worker, 128-129
- bundle branch, 175, 252, 333
- and life expectancy, 421
- and myocardial infarction, 272
- and narrowing of coronary arteries, 255
- preinfarction, 380
- and rehabilitation, 362
- and conduction system, 252-255, 333
- electrocardiographic changes, 207, 333
- and work classification, 333
- Blood, acetylene absorption, 217
- clotting time, 238
- postprandial, 284
- and psychological stress, 295
- coronary sinus, 26, 50, 51
- coronary venous, 21
- arterialization, 94
- and fatty meals, 284, 294
- See also Cholesterol
- Bu
- Calcium ions, and contractions, 14-15
- California cholesterol study, 108
- Industrial Accident Commission 511
- Presumptive Act on heart disease 426
- Workmen's Compensation claims, 423
- and heart injury laws, 435
- Calorie consumption 112, 122-131, 402
- Capillary anastomoses 249
- and atherosclerosis, 241, 245, 248, 249 429
- intramural, 240
- and hemorrhage, 246-250, 291, 296-297
- network, after homograft, 264
- proliferation, and leakage of red cells, 239
- Carbon dioxide, in expired air, 220
- and position, 219 221
- and circulatory congestion, 77-81
- hyperkinetic, 79
- noncardiac, 78
- in congestive heart failure, 78, 82-83, 148, 149
- control in man, 54-58
- and digitalis, 80
- and epinephrine 57, 160
- and exercise tolerance, 56, 145, 209
- in hot and humid environment, 148, 149
- in hyperemia, 56
- in hypertension, 165
- and norepinephrine, 160
- and oxygen consumption, 20, 22-26, 91, 99, 216-217
- and posture, 55, 56, 220-223
- and pulmonary stenosis, 404
- after recovery from coronary thrombosis, 221-222
- and rehabilitation, 216, 220-223
- and salt retention 79
- and stress, 165, 166, 294
- and training, 183-185, 209
- and valvular regurgitation 38-39
- in vasodepressor syncope, 57
- after vasodilating agents, 82-85
- and velocity of coronary flow, 430
- and venous return, 194
- Blood pressure, and aggressive attitudes, 168
- and aging, 127 133-137
- arterial lumen, 248, 249
- and benzodioxane 167
- capillary 248-250
- and heart rate, 62
- ventriculoatrial regurgitation, 34-46
- vessels See Vascular system
- viscosity, 294
- in emotional stress, 165, 166
- and urea nitrogen, 167
- volume, adaptive muscle responses, 258-259
- and beriberi, 78
- and cardiac output, 55, 57, 82
- in circulatory congestion, 77-80, 81
- in congestive heart failure, 77 78, 149-156
- and coronary flow, 50, 430
- and endurance training, 203
- and fitness 189

- Cardiac output (*Contd.*)
 in water retention 79
 and work load, 183, 215
- Cardiac work classification *See* Work capacity classification
- Cardiovascular diseases, 132-139
 hypertensive, 123, 332, 333, 334, 337, 354, 383, 384, 385, 386
See also Hypertensive, Hypertensive heart disease
 incidence, 133
- and aging, 132-139, 384
See also Age factor
 oxygen consumption, hemodynamics, 18-32
- pain, and anxiety, 144
 and exercise responses, 143
- serum concentration, 108
 and emotional conflict, 168, 225
 environmental factors, 108, 225
 and occupation, 109
 and physical activity, 111-115, 167, 201 295
- Chorea 505, 506
- Chromosomes movement 14
- Circulation, and age, 133-139, 389
 and beriberi, 77, 79
 and bradycardia, 509
 and capillary rupture 245, 249
 and cardiac output, 54-58
 collateral, 49 389, 496
 congestion, 76-85
 noncardiac, 77-80
 coronary, 233-241, 243-250, 418, 507, 509
 and anemia, 49 51
 and cardiac energetics, 48-53, 86-97, 248, 430
 and hypoxemia 93-95
 and stress, 90, 92, 243-250, 298-301
 and systolic contraction, 430
 and Workmen's Compensation, 429-431
 and exercise, 109, 168, 216, 217
 hemodynamics, 46, 76-85
 in atherosclerosis, 429-430, 431
 in hot and humid environment, 148-156
 and intrathoracic pressure, 225
 measurement methods, 87, 100, 223
 and metabolic rate, 61-65
 muscles, 158, 268
 during contraction of striated 52
 in myocardial infarction, 389, 430, 431
 in neurocirculatory asthenia, 170-179
 and oxygen consumption, 21, 86-97, 100, 137
 and physical activity, 109, 168 216, 217
 and points of predilection 509
 pooling, in extremities, 194
 and viscera, 216
 and rehabilitation, 216, 217
- Circulation (*Contd.*)
 renal, in congestive heart flow, 78
 and smoking, 294
 and stress, 224, 246, 249
 emotional, 158, 168
 in sudden or unexpected death, 291, 430
 and thrombotic deposition, 245
 tobacco chewers, 294
 velocity, 148, 429-430, 508, 509
 venous, disturbance, 333
 individual differences, 100
- Compensation *See* Workmen's Compensation System
- Conduction system, 251-255, 273, 299
- Congenital heart disease, 325-329, 372, 405, 436
 and absenteeism, 385-386
 and age, 384
 atrioventricular node, 251
 and body types 333
- and work capacity, 123, 326-329, 331-332, 345, 386
 and exercise tolerance test, 162-163, 333
 family history, 332
 and functional capacity, 371
 incidence in industry, 418
 and life adjustment problems 164
 and occupational accidents 385
 and rehabilitation, 336, 352, 358-360, 362, 363
 and sex, 383
- Consolidated Edison Company of New York, 418-419
- Contractile proteins, and movement, 8-16
- Coronary arteries, anastomotic channels, 49, 100
 arteriovenous oxygen difference, 21, 91, 92, 94, 96
 blood flow *See* Circulation, coronary
 diameter alterations, 52, 252, 255, 259, 428, 508-509
 injuries, 428, 488
 insufficiency, after infarction healing, 371-372
 intramural hemorrhages, 233-241, 243-250
 occlusion, 234, 413, 430, 431, 496
 and acute attack, 297-298
 ambulant patients, 413
 and capillary rupture, 246-250
 complete functional cure, 433
 and exertion, 105, 284, 488, 495, 509
 and myocardial infarction, 292, 398
 post-mortem findings, 285, 290-291
 sclerosis, 290-291, 488
 thrombosis, 244, 285, 290-291, 292, 301, 488, 496
- Coronary heart disease 296-301
 and age, 104-106, 107, 259, 283-287, 289 418
 atheromatous, 239, 241, 248, 258, 291-293, 296-297, 429-431
 and atrioventricular block, 255
 and chewing tobacco, 294
 and cholesterol concentration, 108-109, 167-168
 coexisting myocardial infarction, 107, 297-298, 431
 and conduction disturbance, 255, 299, 429-431
 disability benefits 299, 393, 485
 Eastman Kodak Company survey, 418

- Coronary heart disease (*Contd.*)
 and emotional stress, 164, 167-168, 299
 and employment, 105, 118, 286, 289, 296, 313-321, 325-329, 387-388, 420, 485
 exercise as preventive factor, 114
 in Finland, 107
 and functional capacity, 143
 hemodynamic factors, 429-431
 in Indiana farmers, 118
 mortality, 104, 105, 118
 sudden death, 283-287, 288-292, 297 300 430, 497
 pain, and working capacity, 178
 and physical activity, 167, 258, 283, 295, 387-388, 455
 in physicians, 106
 postal and telegraph employees, 105
 professors, 106
 public officials, 106
recovery rates 295
- 332
- 341
- Cortisone, and circulatory congestion, 77, 80
- Degenerative heart disease, 312, 336, 505, 506
- Diabetes mellitus, and arteriosclerotic disease, 271, 371, 384
 and emotional disturbance, 332
 and hypertensive disease, 497
 and myocardial infarction, 271 273 388
 in rehabilitation evaluation, 363
- Dicumarol, 246
- Dietary intake, and atherosclerosis, 112-114, 225, 284, 300
See also Cholesterol
 and capillary fragility, 250
 and coronary disease, 104
 thrombosis, 294
 and cot pulmonale, 104
 and heart failure, 294
 and heart rate, 65
after myocardial infarction, 293
 and physical activity, 109, 111, 112
 in rehabilitation evaluation, 363
- Digitals, in congestive failure, 78 80
 and employability, 319-321
 and rehabilitation, 362, 363
 in rheumatic heart disease, 385
- Digoxin, 143
- Dilatation, 51
 and heart enlargement, 202-207
 and intraventricular pressure, 31
 and peripheral resistance, 158
- Diphtheria, and heart block, 252-253
- Disability, 499-506
 in cardiac cases, 450-451
 claims, 388, 393 394, 510
 death, 450-451
 Utah plan, 424
 and compensation, 393 448-450
 Consolidated Edison study, 419
 criteria for determining, 500
 definitions, 462-463, 500-501
- Disability, definitions (*Contd.*)
 wage-loss concept, 449
 and emotional disturbance, 332
 fear, 433
 and myocardial infarction, 391-393
 period, and prepaid insurance program, 392-393
 and selective placement, 368-374
- Disability Insurance Program, 499-506
 allowance, and age, 504-505
 evaluation guides, 501-502
 "freeze" program, 502
 grace period, 503
 history, 500
 medical report form, 502
 monthly benefits, 500
 and rehabilitation, 503
- and salt intake, 294
- 333, 380
- in neurocirculatory asthenia, 171
 and occupation, 128-131, 384, 385, 388, 389
 pre-employment examinations, 398, 421
 preinfarction, 380
 premature contractions, 380
 in rheumatic myocarditis, 252
 in rheumatic pericarditis, 252
 and sex, 383
- 386, 421
- 437, 509
- and age, 106, 277-279, 297-298, 308, 313-317, 318, 320, 369, 404-406, 409
 future disability, 384, 422
 after Altro workshop, 319
 and angina pectoris, 314, 318, 319-320, 371, 372
 and anxiety, 317, 318

Employment of cardiac (*Contd*)

- attendance, 393, 399-422
 - See also* Absenteeism
- Baton Rouge Refinery, 368-374
- and causal relationships, 388, 393, 394, 405, 426-431, 464, 508-509
- and terminology, 491
- Consolidated Edison Company of New York, 448, 450, 457
 - and cost of compensation insurance, 442-443, 448, 450, 457
 - and Second Injury Laws, 435-437, 508
- course of continued, 420
 - advancement in position, 394
 - changes and modifications, 398, 407-408, 409, 419
- 369, 398, 400-409, 421
 - exclusion attitudes, 419, 421, 422, 507, 508
 - factors for, 312, 315-321, 334, 343-406
 - and limited in-plant mobility, 422
 - motivation, 146-147, 316, 317, 318, 335
 - social, 335-407, 409
 - and time of last work, 317-318, 373-374
 - financial considerations, 317, 318, 406, 409
 - Ford Motor Company, 397
 - Gillette Safety Razor Company, 383-399
 - in Great Britain, 400-409
 - and group insurance plans, 423
 - identification of workers, 368
 - and job classification, 123
 - See also* Occupation
 - and medical supervision, 412-508
 - therapeutic, 319-321
 - and mental ability, 407, 409
 - morale effects on fellow workers, 386
 - noncardiac reasons, 409-411
 - performance rating, 386
 - personal attributes, 406, 407-409
 - and physical fitness, 318-334, 335, 405
 - improvement, 337-338, 410
 - potential compensation claim, 309-466, 508
 - premature retirement, 388
 - and previous work experience, 317-318
 - and production profit, 386, 422
 - prospects for, 406
 - and psychological outlook, 135-316, 518
 - resettlement, 404-406, 409
 - See also* Rehabilitation
 - safety, 279-280, 286-289, 316-317, 385-386, 507, 508, 509
 - selective placement, 368-374, 383-399, 432-437, 509
 - and stability of previous history, 405-406, 407-409
 - and vocational characteristics, 334
 - See also* under specific type of heart disease
 - Endocarditis and conduction system, 251, 255
 - and mortality in Indiana farmers, 118
 - Endothelium, in atherosclerosis, 238, 239-241, 429
 - Environment, in heart disease incidence, 103-107-109
 - humid, 185-193, 211-214, 225, 397
 - and hot, 148-156
 - See also* Temperature
 - environmental
 - and oxygen consumption, 186-188
 - and restrictive activity, 397
 - and trauma concept, 427
 - and work classification, 372
 - duration, 68, 144-145, 181, 183, 189
 - and recovery, 145, 181-183, 189
 - time, and environment, 190
 - and training, 184, 207, 209
 - electrocardiographic complexes, 144-145, 164-165
 - and heart, 159, 199-209
 - rate *See* Heart rate
 - and hypertension, 167
 - and longevity, 200-202
 - and neurocirculatory asthenia, 160-162, 164
 - after occlusion, 109
 - and oxygen consumption, 137
 - debt, 224
 - 143-145, 224
 - and stroke volume, 137
 - and sugar intake, 64
 - tolerance, 160
 - and cardiac output, 56-145, 209
 - and functional capacity, 143, 145-146, 403
 - and hypertrophy, 204
 - and individual differences, 114-115
 - and lack of motivation, 115
 - and occupation, 128-129, 207
 - patent ductus arteriosus, 162, 163
 - validity, 114-115
 - and work classification, 333
 - and vascularity, 109
 - See also* Physical activity
 - 214
 - Federal Social Security Programs, 499-506
 - Femoral artery occlusion, 49
 - Fenn-Hill hypothesis, 4
 - Fibril, contraction, 15
 - Fibrillation, auricular, 128-129, 372
 - in functional heart disease, 175
 - and rehabilitation, 362, 363
 - in rheumatic heart disease, 372
 - ventricular, 498
 - and fatal arrhythmia, 430
 - after iced beer, 293
 - and myocardial ischemia, 298
 - and Workmen's Compensation, 428
 - Fibrin, in atherosclerosis, 237, 240
 - Fibrolasts, amnion, contraction, 9-11, 99
 - and conduction system, 251
 - Fibrosis, 281, 291, 292, 429

- Finland, myocardial infarction incidence, 108
- Fistula, arteriovenous, 77, 80
and cardiac output, 56-58
- Block test, 225, 228
- Florida, heart injury laws, 435
- Force platform, 196-198
- Ford Motor Company, 397
- Galveston District Heart Association, 311
- Gangrene, arteriosclerotic, 271, 272, 273
- Garment workers, New York City, 388-391
- Gillette Safety Razor Company program, 383-399
- Graft, musculofascial, vascularization, 257-268
- Great Britain, 105, 400-409
Social Medical Research Unit, 120
- Greater Peoria Heart Association, 311
- Hawaii, cholesterol study, 108
Workmen's Compensation laws, 411
- Health Insurance laws, and workmen's compensation, 461, 510
- Heart, arrest, 298, 301, 428, 430
beat, 182, 183, 190
premature, 173, 333
block *See* Block
dilatation, 31, 51, 202-207
distention, 216
electric shock, 427
and electrolyte imbalance 497
flutter, 175
function, 31-32, 80-92, 93, 98, 199, 200, 207, 494-495
disorders, 170-179
isolated supported dog, 19-22
and Laplace's law, 74, 100
muscle *See* Muscle, heart
right coronary preponderance, 49
shape, 74, 100
size, 74, 100, 167, 183, 200-207, 320, 321, 385
and aging, 133
and employability, 318, 372
and failure, 77, 372
and myocardial fiber tension, 31
in neurocirculatory asthenia, 171, 174
and rehabilitation, 362, 363
and tension, 75
and Starling's law, 82-84, 85, 203
strain, 193-195, 213, 224, 481, 485, 498
definitions, 427, 494, 495
individual differences, 494
time interval for first manifestation, 497-498
stroke volume *See* Stroke volume
structural defects *See* Congenital heart disease
and training, 60-62, 68, 183-185, 199, 200-207
weight, 89, 90, 92, 93
after failure, 272
work, 48-53, 60-62, 68, 73, 141-143, 183-185, 199, 200-207, 248, 430
bed-bound patients, 222
and braking forces, 198
capacity *See* Work capacity classification
chair position, 222
in decompensation, 152
and humidity, 148-156, 185-193
in hypoxemia, 93
nervous and humoral factors, 100
and oxygen consumption, 86-97
vasoregulatory asthenia, 171
See also specific conditions and diseases
- Heart failure, and angina, 371
in arrhythmias, 371
and arteriosclerotic heart disease, 371, 372, 384
and blood pressure, 272
congestive, 51, 76-85, 153, 271, 371, 372, 384, 385
- Heart failure, congestive (*Contd.*)
and disability under Old-Age and Survivors Insurance, 503
and overexertion, 496
recognition, 76-77
and Starling's law, 82
and Workmen's Compensation, 427, 450
and coronary blood flow, 51
and employment, 319-321, 374
in hot and humid environment, 148-156
in hypertensive heart disease, 83-84, 271, 371, 372
in luetic heart disease, 371
and myocardial infarction, 271, 273, 371
recovery, 78-80, 81, 295
in rehabilitation evaluation, 361, 363
and renal hemodynamics, 78
rheumatic heart disease, 371, 372, 385
- Heart rate, and age, 71-72, 185
anticipatory rise, 173, 180, 226
and Atropine, 56
after blood infusion, 40, 42
and blood pooling in lower extremities, 194
and blood sugar, 64
and body size, 72
and cardiac output, 55, 56
and cardiac stress, 75, 225
and coronary flow, 89, 90, 91, 430, 509
during digestion, 62
electrocardiographic complexes, 164-165
and energy expenditure, 122-131
cumulative effect, 68, 181
in exercise, 56-58, 60-62, 68, 168, 180-195, 207-208
and sympathetic stimulation, 71, 72-73
and heat stress, 63-70, 186-188, 194-195, 225
and acclimatization, 192
in hyperemia, 56
individual differences, 100
measurement, force platform, 196-198
and mephentermine sulfate, 39, 40
in mitral regurgitation, 38, 39, 43-44
and oxygen consumption, 28-32, 90, 91, 186-188
and physical fitness, 192
and training, 60-62, 68, 183-185
postural studies, 56, 65, 192, 194, 219
and rectal temperature, 70
regulation, 60-73
and relative humidity, 185-192
resting, or steady state, 60-72, 68, 73, 161, 181-182, 183
and sexual excitement, 294
and ventricular pulse pressure, 30
and work load, 180-195, 198
- Hematoma, 245-246
- Hemodynamics, circulatory, 76-85
and adaptive process, 168
and cardiac function, 40, 42, 77, 81-85, 165
in occlusive disease, 430, 431
and valvular regurgitation, 34-46
in congestive heart failure, 78-80
and Arionad, 82-83
and digitalis, 78-79
and Starling's law, 82-84
in coronary heart disease, 429-431
myocardial, 80-81
in oxygen consumption, 18-32
renal, 78
- Hemorrhage, intramural, and atherosclerosis, 233-241, 246, 296, 428, 429-430, 431, 508-509
capillary origin, 246-250, 291, 296-297
and coronary occlusion, 243-250
and hemosiderin pigment, 233, 301
stenosing effect, 245, 248

- Hemorrhage, intramural (*Contd*)
 and sudden death, 247, 291, 300-301, 430
 and thrombus, 243-246, 297, 300
 and Workmen's Compensation, 428, 508-509
- and heart work, 185-193
- Hyalinization, and atherosclerosis, 235
 grading, 235
- Hyperemia, and cardiac output, 56
- Hypertension, and accidents, 385
 and age, 384
 in congestive heart failure, 83-84
 Consolidated Edison survey, 418
 and coronary blood flow, 51
 and Disability Insurance Program, 505
 and emotional disturbance, 165, 332
 and employment, 314, 318, 320, 345
 and absenteeism, 385-386
 and exercise, 167
 family history, 332
 and functional capacity, 371
 and intracapillary pressure, 247, 249
 and myocardial infarction, 388
 fatality rate, 378-379, 380
 healed, 271-273
 and occupation, 104, 384
 in Russia, 109
 and sex, 383
 and Workmen's Compensation, 428
- Hypertensive heart disease, 507
 and acute attack, 296
 and age, 354, 384
 and cardiac strain, 495
 conduction system, 251, 252
 at Consolidated Edison Company, 418
 disability benefits, 393, 505
 dyspnea, 371
 at Eastman Kodak Company, 418
 and employment, 309, 313, 314, 326-329, 331-332, 334, 335, 337, 345, 360, 422, 496
 and energy expenditure, 127-128
 and failure, 372
 congestive, 77
 and functional capacity, 371
 mortality, 104, 118
 and race, 506
 and rehabilitation, 352, 354, 358-360, 362, 363
 and sex, 505
 and work performance rating, 386
- Hyperthyroidism, 51
- Hypertrophy, 49, 207
 and exercise tolerance, 202-204
 and healed myocardial infarction, 271-273
- Hypoplasia, 251
- Hypotension, exertional, 143, 145
 hemorrhagic, 51, 235
 and occupation, 109
- Hypothermia, and oxygen consumption, 51
- Hypothyroidism, 51, 273
- Hypoxemia, and cardiac oxygen consumption, 93-95
- Illinois, work classification unit survey, 341
- Indiana, heart disease in farmers, 117-121
- Industrial Accident Commission, California, 511
- Industry and heart disease (*Contd*)
 comprehensive security program, 513
 and compensation system. *See* Workmen's Compensation System
 education need, 420-421
 and energy cost of operations, 402
 environmental control, air-conditioning, 372
 limited in-plant mobility, 422
 incidence, 417-418, 507-508
 insurance, 446, 483, 496, 513
 costs, 442-443, 448, 450, 457
 group, 423
 health and sickness, 422
 medical care, 443-444
 and rehabilitation, 445, 454-459, 507, 509-510
 self, 458
 waiver of premium provision, 500
 Workmen's Compensation, 422, 423, 426, 435, 438-453, 510
 and physician, 420-421
 pre-employment screening, 396, 418, 496
 protection under second injury laws, 435, 508
 selective placement, 368-374
 and Utah Plan, 483
 for evaluating, 487-498
See also Employment of cardiac, Occupation, Workmen's Compensation
- Injury, chest, 428, 481, 494
 concept, 427
 definitions, 462, 494, 510
 employers' liability for, 422, 423, 426, 435, 439-440, 451, 510
 and "second injury" laws, 435, 508
 intrathoracic, 428
 penetrating wounds, 427, 494
 and permanent total disability, 435
 and pre-existing defect, 428, 492-493
 and traumatic heart disease, 426, 463-464, 488, 494
 Utah Medical Panel System, 482-498
See also Accident
- Institute for Crippled and Disabled, 458
- Institute for Physical Medicine and Rehabilitation, 458
- Insufficiency, aortic valvular, 372
 coronary artery, 433
 after infarction healing, 371-372
 mortality, 104, 430
 mitral, 385
 myocardial, 389-390
 and Workmen's Compensation, 428
 rheumatic, 155, 385
 ventricular, and congestive failure, 155
- Intima, hemorrhage and coronary occlusion, 243-250
 and histogenesis of atherosclerosis, 233-241, 297, 429
- Iowa Heart Association, 341
- Irradiation, and homograft-host interactions, 266, 268
- Ischemia, and capacity for effort, 403-404, 405
 coronary, 252, 253
 myocardial, 298, 300-301
 and oxygen consumption, 51
 survival, and work return, 411
- Italy, heart disease incidence, 108
- Japan, cholesterol levels study, 108
- Kentucky, heart injury laws, 435
- Kessler Institute of Rehabilitation, 458
- King's adaptation of Bernoulli's principle, 248
- Knees, energy for bending and straightening, 198
- Kyushu, cholesterol study, 109

- Laplace's Law, 31, 74-75, 160
 Laro's force platform, 156-158
 Liberty Mutual Insurance Company, 458
 Lipids, blood, 167
 and atherosclerosis, 168, 235, 239, 301
 and heparin, 284
 pools and intramural hemorrhages, 231, 241
 Lipoprotein, 113
 Liver, enlargement, and rehabilitation, 361, 363
 Lockheed Aircraft Corporation, 419
 Longshoremen's and Harbor Workers' Compensation Act, 411
 Los Angeles County Heart Association, 341
 Lumen, atherosclerotic changes, 237, 239, 249
 429-430
 and intramural hemorrhage, 238, 241, 244
 occlusion, and sudden death, 300-301
 Magnesium, and contractile proteins, 9-10
 Maryland, independent examiner system, 510
 Massachusetts, Cardiac Work Classification Unit
 in Boston, 311-321, 341
 and Workmen's Compensation, 422-423, 441
 Massachusetts Heart Association, 341
 Master's exercise test, 122, 128-131, 159, 333
 Mayo Clinic study, 417-418
 Mephentermine sulfate, 38-40
 Mercurials, 320, 362, 363
 Metabolism, and body temperature, 67-71
 electrolyte imbalance, under Utah Law, 497
 and energy costs, 215, 402
 in functional heart disease, 175
 and heart rate, 60-62, 66
 of muscle cell, 257-268
 and physical activity, 60-62, 215-217, 223
 and position, 217-223
 Metazoa, contraction cycle, 15
 Minnesota, heart injury laws, 435
 Missouri, heart injury laws, 435
 Mitral valve, 51
 endocarditis, 255
 and regurgitation, 34-46, 99
 in rheumatic heart disease, 372, 385
 stenosis, 77, 79, 163
 Mortality, 450-451
 at Alto, 366-367
 arrhythmia cardiac standstill, 430
 arteriosclerotic heart disease, 338, 388
 coronary disease, 104-105, 118, 290-291, 292,
 300-301, 488, 498
 cor pulmonale, 337
 and endocarditis, 118
 epidemiological conclusions, 104-105
 in hypertensive heart disease, 104, 118
 Indiana farmers, 117-121
 and myocardium, 104, 105, 118, 279-280, 292,
 301, 378
 in rheumatic heart disease, 118
 See also Sudden death
 Motivation, and employment, 316-318, 335
 and functional capacity, 146-147, 227
 Movement, and contractile proteins, 8-16
 Muscle, adaptive responses to work, 258
 arteriovenous oxygen difference, 208
 blood flow, 52, 158
 contractile process, 32, 52
 active relaxation cycle, 5, 98
 and adenosine triphosphate, 9-16, 99, 518
 in cold- and warm-blooded animals, 13
 energetics, 3-6
 Hill's quick-release phenomenon, 13-14
 and mersalyl, 9-12, 16
 passive relaxation cycle, 4
 phosphocreatine breakdown, 30
 contractile process, C₁ and
 pyrophosphate, 14
 tetanus, 5, 8
 in delta state, 4, 68
 energy release, 5, 9-16, 125
 and exercise tolerance, 167, 210
 See also Exercise
 Fenn-Hill hypothesis, 4, 5, 6
 force-velocity relation, 4, 58
 in tetanus, 6
 heart, 70, 98
 cardiac regulating mechanisms, 10
 host-hemolysis interactions, 257, 268
 and hypercholesterolemia, 168
 metabolism, 17
 mechanical activity, 258
 necrosis and healed infarction, 301
 oxygen consumption, 209
 resting stretched beyond resting length, 5
 rigidity, 16
 and skeletal system in absterium, 356
 in disability claims, 332
 thermal responses, 3-6, 9-10, 11, 98, 265-268
 work, 158, 185-192, 268
 discussion, and response, 159
 and lipid metabolism, 167, 168
 Myocardial Infarction Register, 388
 Myocardium, and bundle branch, 252, 254, 255
 and conduction system, 251-255
 incidence in industry, 418
 Myocardium, atherosclerotic disease, 292, 297
 and work, 301
 autopsy findings, 269-273, 280-281, 290-291
 circulation, 48-53, 251-255
 collateral, 389
 contractility, and mural regurgitation, 46
 contusion, 431, 494
 in evaluation for rehabilitation, 215
 focal fibrosis and sudden death, 290-292
 function, and congestive failure, 80-82
 in hot and humid environment, 150
 hypertrophy, and work load changes, 239
 infarction, 269-273, 371-372
 and accident, 428, 488
 and age, 108, 270-271, 273, 276-277, 315,
 318, 320, 376, 379, 381, 384, 388-389,
 390, 392-393
 ambulant patients, 390-391
 angina, 271, 273, 389-390
 apical, 270
 asymptomatic, 496-497
 and awareness, 269-273, 297, 390-391, 496
 and blood pressure, 269
 cardiac output, 57
 and cerebral thrombosis, 271-273
 classification, 279
 and coronary disease, 290-292, 297-298, 398,
 431
 and diabetes, 271, 273, 388
 diagnosis, 271-273, 280-281, 297-298, 389-
 391
 "disability period," 391-392
 at Du Pont Company, 375-382
 and education, 318
 and emotional disturbances, 430
 and employability factors, 315-321, 371
 in employers, 278-280
 and exertion, 430, 488, 496
 in garment workers, 387-394
 geographical distribution, 376
 healed, 269-273, 300, 371, 390
 and high-fat meal, 294
 and hypertension, 271-273, 378-379, 380, 388
 incidence, 170, 269-273, 274-282, 380-382,
 384

290-293,

37

multiple, 384

and occlusive disease, 290-292, 398

and occupation, 277-279, 380-382, 488

onset during working hours, 371

painless, 390-391

and psychiatric status, 270-271, 273

and race, 270-271, 273

recovery, 293, 374, 376, 378-380, 392-393, 411

recurrence, 318, 398-399

and season of year, 376-377, 378

and sex, 270-271, 273, 276-277, 376, 388-389

and sudden death, 290-293, 297-298, 430, 497

time before work resumption, 373-374, 387

wives of employers, 278

work capacity after, 123, 127, 319-321, 333, 372, 384

years of work since, 374, 384

injury, 428, 431

ischemic changes, 252, 298

and occupational therapy, 222-223

oxygen consumption, 18-32, 89, 90, 97

right ventricle congestive failure, 148-156

toxins, and muscle work capacity, 258

tumors, and atrioventricular node, 255

vascularization, 257, 267

and exercise, 109

Myofibrils, contractile proteins, 9

National Association of Claimants' Compensation Attorneys, 456

National Employment of Physically Handicapped program, 458

Nebraska heart injury laws, 435

Nephritis, and circulatory congestion, 78, 79, 80

Nephrosis, 77, 78

Neurocirculatory asthenia, 170-176, 178

Neurosis, 352

cardiac, 170-176, 374

and substandard work status, 373

compensation and absenteeism, 470

35

New Jersey Heart Association, 341

New York City Bellevue Hospital Work Classification Unit, 305-310

Ladies' Garment Industry, 388-394

New York Heart Association, 420

functional classification, 403, 408

night clinic for cardiac workers, 508

New York State, 435

accident within meaning of Workmen's Compensation Law, 427

compensation boards, impartial experts, 511

court plan for compensation litigation, 477, 478, 510

heart disease laws, 508

"second injury," 435, 436

one-unit plan of pneumoconiosis and pulmonary diseases, 511-518

Workmen's Compensation, 422, 423, 441, 444, 455

hearings, 452, 510

pretrial conference, 477, 478, 510

Nicotine, 294

Nucleoside triphosphate, 9-12

Obesity, 109, 319-321, 389

Occlusion, of arterial lumen, 284

atherosclerotic, 285, 290-291, 496

agricultural worker, 335, 342, 351

and arteriosclerotic heart disease, 119-121

bakers, 351

building industry workers, 351

bus conductors, 420

bus drivers, 351, 421

businessmen, 107, 351

butchers, 351

chefs, 351

clerical, 109, 113-114, 119-121, 278-280, 335,

342, 363, 372, 373, 380-382, 384

communication, 335

construction, 335

contract, 335

and coronary disease, 105, 143-147, 224, 286,

335

insurance, 442

and workmen's compensation statutes, 461

doctors, 109

domestic service, 335

electrical workers, 351

employers, 278-280

and energy cost, 127, 142, 192, 402, 495

executives, 107, 109

and exercise tolerance, 207-208

factory workers, 122-131, 351

farmers, 109, 117-121, 142, 211-214, 335

finance, 335

fishery, 342

food industry workers, 351

foremen, 107

garment workers, 351, 388-394

government, 335

and heart attack, and causal relationship, 325,

390-391, 495, 509

horticulture, 335

housewife, 325, 335

and hypertension, 104, 109, 384

and hypotension, 109

industrial, 193-195

heavy, 113-114

insurance, 335

in Kyushu, 109

laborers, 119-121, 289, 380-382

skilled, 325, 335, 342, 372, 373, 380-382,

384

unskilled, 317, 325, 335, 342, 372, 373, 384

mail carriers, 420

in Malmö, Sweden, 277-280

managerial, 325, 335, 342, 351

- Occupation (*Contd.*)
 manufacturing, 335
 mechanical workers, 351
 miners, 109, 335
 and myocardium infarction 277-280, 318, 380-382
 in New York, 289
 in Norway, 107
 officials, 335
 operatives, 119-121
 personal service, 335
 and physical fatigue, 193-195
 postal workers, 420
 and pre-existing pathology, 509
 professional 107, 119-121, 289, 335 342, 351, 380-382
 protective service, 335
 real estate, 335
 after rehabilitation 351 356-357
 and responsibility 381 382
 retail, 335
 in Russia, 109
- 382
- 1-195
- 224, 298
- students, 342
- supervisory, 372 374 380-382
- switchmen, 114
- taxi drivers 351
- teachers 109
- technical, 372 373
- telegraphers, 109
- telephone operators, 109
- transportation 335
- truck drivers, 351
- and vulnerability 373
- white collar workers, 289
- wholesale, 335
- Ohio, 445
- 330-339
- 36
- in congestive heart failure, 78 79
 coronary, 50, 94, 96
 and exercise tolerance, 208
 muscle, 209
 in rehabilitation 216
 after therapy, 83, 91, 96
 concentration in expired air, 218
 consumption, 18-32, 86-97, 216
 and athletic performances, 199
 and braking forces, 198
 and cardiac efficiency, 91, 136
 in cardiac factory worker, 128
 after circulatory congestion recovery, 81
 in congestive heart failure, 78
 and coronary flow, 94
 and energy requirements, 75, 81, 100, 141-149 225, 402
 and environmental temperature, 67-71, 185-192
 exercise, 137, 225
 and external efficiency, 21, 26 31-32
 hemodynamic determinants, 18-32
 and hypoxemia, 93-95
 myocardial, 50, 51, 87
- Oxygen, consumption (*Contd.*)
 and position, 217-223
 and rectal temperature 70-71
 in rehabilitation of cardiac, 216
 and relative humidity 185-192
 and stress, 51
 tension-time index, 23 27, 99 228
 of trained athlete, 68
 and ventilatory equivalent 128
 and work capacity, 114, 177, 225
 saturation, and posture, 65
- Panel system in compensation cases, 482-486
 491-498 510
- Parametria, trichocysts, 15
- Pennsylvania, Cardiac Work Classification Unit
 322-329 341
- Pericarditis, and conduction system, 77, 79, 251, 252, 255
 tuberculosis, and rehabilitation 352, 358-360
- Work
 111-115
- 167-168, 295
- in causal relationship 431, 481
 and acute attacks, 296, 495, 508, 509
 and effort, in bending knees, 198
 definition, 494
 and energy cost 5-6, 91, 125
 for specific activity, 141-143 402
 and fitness, assessment, 140-149, 183 228, 369, 370, 403, 434
 at Alto workshops, 348-367
 and electrocardiographic changes 128-131
 and emotional stress, 158-169 222
 and fatigue, 143, 193-195 198
 and heart responses, 60-62, 68, 183, 192 199, 200-207 226
- 176
- capacity
- Pressure, and aging, 133-137
 aortic, 28, 29, 38 39, 43-44
 and oxygen consumption, 20-22 87
 and antigravity suit compressions, 55
 and Atropine, 56
 arterial *See* Blood pressure
 atrial, 19-22, 38-39, 43, 46, 99
 and blood volume 55
 and posture, 56
 Bernoulli's principle, 247-248
 and cardiac output, 28, 55
 and circulatory congestion, 77, 78 81
 diastolic, 20, 29, 41, 77, 81, 127, 133-137 269
 and intimal capillaries 247
 and ventricular work, 39 40, 46, 99
 filling, 37, 39, 43, 46, 82-83
 in rehabilitation, 215, 216
 intra-abdominal, 294

Pressure (*Contd.*)

- intrathoracic, for farmer, 213
- in isolated supported dog heart, 20
- King's adaptation of Bernoulli's principle 248
- and mephentermine, 38-40
- and myocardial infarction, 269
- and oxygen consumption, 26, 28-32, 100
- pulmonary artery 50 77, 81, 99
- systolic, 26, 28, 30, 77 127, 133, 145, 169
 - and emotional excitation, 215, 294
 - and stroke volume increase, 28-29
- in valvular regurgitation, 37-46 99
- ventricular, 20, 37-39 40 46 77, 81, 99, 133-134 216

Pulmonary artery, 361

- conus, 372
- disease and emotional disturbance, 332
 - work classification unit survey, 345
- embolism, 272
- engorged 372
- oxygen consumption 21
- stenosis, 51 272, 404
- and Workmen's Compensation 427 428

Pulse rate, 216

- champion skiers, 204
- and emotional excitation, 162, 294
- and energy expenditure 128-131
- environmental response 148, 193
- and exercise tolerance, 161 207
- and fatality rates in myocardial infarction, 379-380
- and fitness, 177 183 192 207
- and heart rate recovery 192 194
- individual differences 114
- in neurocirculatory asthenia, 161
- and position 220, 221
- and pressure-volume cycle, 153
- and rehabilitation activity, 216, 221
- and stroke volume ratio, with age, 125
- and work load 193

Pyrophosphate, and contraction, 14

Race and coronary artery disease, 506

- and myocardial infarcts, 270-271, 273
- and period of disability 392, 506
- and work classification 323-324

Regurgitation valvular, hemodynamic effects, 34-46 99

Rehabilitation of cardiac, and age, 351 354, 357 358, 363

- at Alto workshops, 348-367
- and angina, 362, 363
- in arteriosclerotic heart disease, 352, 354, 358-360, 362, 363
- and blood pressure 216 361
- circulation, 216, 217
- Committee of American Heart Association 419
- and cor pulmonale, 336
- and cost of compensation insurance, 442
- counseling and social work, 413
- and education level, 350-351, 355, 357, 363
 - of attending physician 420, 508
- emotional 336, 338
- and employment, 336, 338, 359, 360, 432-437
 - and job placement, 419-420, 509
 - range of occupations, 356-357
- Esso Baton Rouge Refinery, 368-374
- and ethnic background 351, 357, 358, 363
- factors for successful, 322-329, 352-353, 357, 361, 363, 434, 436
- Federal-State vocational program, 456-457, 458

Rehabilitation of cardiac (*Contd.*)

- and functional capacity, 215-223, 352-354, 356 357, 358, 359, 361, 363, 378, 501-502
- in Great Britain 400-404 420

363, 367

355 357 363

- psychosocial, 179, 225, 350, 355
- rheumatic heart disease, 352, 354, 358-360, 362, 363 406

305-310, 420

Cleveland, 420

Philadelphia 322-329

- and Workmen's Compensation, 441, 454-459, 507-513

Remploy Ltd 400

Respiration, 50

- in coronary attack, 390
- and emotion, 114
- and energy expenditure, 124, 128-131
- in neurocirculatory asthenia, 171-179
- neurosis, and work capacity, 178
- orthopneic position, 222
- in rehabilitation, 216, 217-218

Rheumatic fever, 104, 428 505, 506

Rheumatic heart disease, 352 405

- and age, 384
- and body types, 333
- capacity assessment, 403-404
 - after employment, 337
- and cardiac strain, 495

- and employment, 309, 313, 326-329, 371, 496
 - and absenteeism, 385-386
 - after Alto discharge, 360
 - factors for, 325, 334
 - performance rating, 386
 - and exercise tolerance test, 333
 - family history, 332
 - and heart size, 385
 - industrial risk, 385
 - and mortality in Indiana farmers, 118
 - and occupation, 118, 384
 - and race, 506
 - and rehabilitation, 336, 352, 354, 358-360, 362, 363, 496
 - and sex, 383, 505
 - and strain, 427

54-

total,

5

- Rheumatic heart disease (*Contd*)
 and Utah Panel Plan, 497
 and work capacity, 123, 128, 403
 classification, 326-329, 331-332, 345, 372
 Rhode Island, heart injury laws, 435
 Russia, hypertension, and occupation, 109
- San Francisco Heart Association, 341
 Serum, cholesterol levels. *See* Cholesterol
- Stress (*Contd*)
 emotional, 158-169, 222, 295, 332
 and blood pressure, 71-73, 90, 130, 162, 167,
 246, 291, 295
 in congenital heart disease, 162-165, 167
 and electrocardiograms, 164, 165
 and exercise, 114, 158-169, 160-165
 and neurocirculatory asthenia, 160-162, 164,
 170-176
 respiratory response, 114
 and sleep, 124
 and energy expenditure, 122-131
 environmental, 186, 194, 224, 225
 cardiac farmer, 117-121, 211-214
 of intracapillary pressure, 246-250
 myocardial ischemia acute, 297-298
 nonoccupational, 293-295
 occupational, 104, 122-131, 192, 485
 industrial liability cases, 293-295
 and workmen's compensation, 298-299
 and oxygen consumption 90, 225
 and pain, 298
 physical, 129, 168, 192, 194, 224, 246, 283-287
 294, 295, 298
 ballistic time, 213-214
 and circulation studies, 176-179
 in factory workers, 122
 handball playing, 293
 and respiratory rate, 128, 294
 sexual, 293, 294
 social, 225
 suburban living 299
 sports 294
 and sudden death, 283-287, 290, 291, 293, 298
 Stroke volume, and aging, 133, 135, 137
 of athlete, 61-62, 183-185
 and atropine, 56
 after blood infusion, 40, 42
 and cardiac output, 55, 56
 in congestive heart failure 149, 153
 emotional response, 158, 159, 162, 163, 165, 166,
 167
 and environment, hot and humid, 148, 149, 153
 and exercise, 56-58, 159, 168
 discussion, 158
 in hyperemia, 56
 in hypertension and stress, 165
- Sudden death 497
 in coronary disease, 290-291, 292, 300-301
 occlusion, 283-287, 297-298, 430, 497
 and intramural hemorrhage 247, 291, 300-301,
 430
 Medical Examiner's Law in New York City, 288
 and myocardial infarction 290-293, 297-298
 430, 497
 and occupation 289, 497
 and physical activity, 286, 290
 and stress, 283-287, 290, 291, 293, 297-298
 and thrombosis, 297-298, 430, 496-497, 498
 and workmen's compensation, 288, 298-299
- Conduction system 247, 251, 252
 and employment, 334, 337, 405
 and functional capacity, 333, 345, 371, 372
 and gummatous myocarditis, 252
- Stress adaptation-mechanism for, 51, 91-92, 158
 169
 and cardiac output, 165, 166, 222, 294
 causal relation to heart disease, 293-295, 431,
 485
 time relation, 497
 daily of normal activities, 222, 294
- Sinus rhythm, 128-129, 172, 333
 Skung, ex-champions, 200-206
 Sleep, and angina pain, 496
 and emotional stress, 124
 fatal attack during 290
 and coronary thrombosis 488
 heart effort during 494
 Smoking, 51, 293, 294
 Social Security program, 394, 465, 499-506
 Somatotypes, in heart disease 332-333
 South Africa, cholesterol study, 108
 Spain, cholesterol study, 108
 Standstill, and fatal arrhythmia 430
 and ischemic myocardium 298
 and sudden death, 301
 Starling's law, 82-84, 85, 203
 Stenosis aortic, 155, 385, 404
 and work capacity, 128
 atherosclerotic, 249, 371
 of lumen, 245, 248
 mitral 385, 404
 and blood flow 77, 78, 251
 and exercise tolerance, 162-163
 pulmonary, 251, 404
 pyloric compensability and expert opinion 472-
 474
 in rheumatic heart disease, 155, 385
 tricuspid, 77
- Stress adaptation-mechanism for, 51, 91-92, 158
 169
 and cardiac output, 165, 166, 222, 294
 causal relation to heart disease, 293-295, 431,
 485
 time relation, 497
 daily of normal activities, 222, 294

- Syphilitic heart disease (*Contd*)
 and heart size, 372
 incidence in industry, 418
 and rehabilitation, 336
 and Workmen's Compensation, 428
- Tachycardia, 159
 and fatality rates in myocardial infarction, 379-380
 in functional heart disease 175
 in neurocirculatory asthenia, 171
 paroxysmal, 173, 333, 371
 in recumbent position, 57
 and rehabilitation, 362
 sinus, 128, 172, 333
 and starvation, 62
- 70-71
- in congestive failure, 148-156
 and muscular work, 185-192
 heat stress, 148-156, 211-214
 and host-graft interactions 266
 and muscle, 3-6
 contraction, 9-10 13
- 25
- 341
- 238
- 97, 428,
- and exercise, 109, 403-404
 and exertion, 291 488, 496, 497 498
 fatal sleep, 488
 and myocardial infarction 292 497
 and myocardial ischemia, 298
 and occupation 301, 381
 and pain 496
 and intramural hemorrhage, 235 236, 248, 239,
 243-246 297, 300, 429
 luminal 233-241 430
 recurrent, 240
 secondary, and stress 246
 standardization of terminology 299
 and sudden death 291, 297-298 430 496, 497,
 498
- Training, 200-208
 and adaptation 158, 209 226
 and blood volume 203
 and exercise tolerance 184 207, 208, 209
 and heart work 60-62 68 181-185 199, 200-
 207, 209
- Trauma, concept range, 427, 194
 definition, 494
 and heart disease, 426, 463-464, 488, 494
See also Injury
- Tricuspid valve, atresia, 251
 stenosis, and blood flow 77
- Trimethaphan camphorsulfonate, 82-83
- Tuberculosis, 105
 disability, 499
 preplacement and periodic examinations, 398
 and rehabilitation 348 349 352 358-360 362,
 363
- United States Department of Labor, Bureau of
 Labor Statistics 419
- University of Buffalo, New York, 341
- 512
- 189
- evolution, 488-490
 heart disease claims, 393, 424, 497
 compensation criteria, 464, 510
 heart injury laws, 435
 second injury fund, 484
 mechanics, 484, 491-493
 procedure for medical data, 492-493
 report and hearing procedures, 489-491
 appeal from 493-494
 formal findings and conclusions, 484, 489
 selection of medical panels, 484, 489, 491
- Valsalva maneuver, 202, 225
- Valvular heart disease, 296
 and congestive heart failure, 77
 hypertensive, Eastman Kodak Company survey,
 418
 incidence, 170
 and intercoronary collaterals 49
 mortality, and workload, 105
 and muscle work capacity, 258
 obstructive lesions, 404
 rheumatic, healed phases, 252
See also Aortic valve; Mitral valve
- Vascular system, conduction, 251-255, 273, 299
 disease *See* specific diseases
- in emotional stress, 165-167 168
 and epinephrine 160
 and exercise, 138, 139, 145, 158, 167
 in hypertension, 165, 167
 and mitral regurgitant flow 36-37
 and norepinephrine, 160
 and oxygen consumption, 21
 peripheral 39, 40, 42, 50, 57 133, 135, 138,
 145, 158, 160, 165-167, 168, 294
 pulmonary, 50
 and stress, 165
- 82-85
- 249,
- 429
- Ventilation, and heart rate in athlete 68
 and metabolic rate, 61-65
 pulmonary, in cardiac factory workers, 128
 in muscular exertion, 158
- Ventricle, aneurysm, 333
 after Arfonad, 84
 in congestive heart failure, 153
 contusion, 428
 electrocardiograms 145
 preinfarction, 380
 fibrillation 298
 and sudden death 301
 filling, 58
 in recumbent position, 57
 function, 153
 and myocardial oxygen consumption, 20-36
 in open-chest dog, 20
 in ventriculoatrial regurgitation, 37-46
 in hot and humid environment, 148-156
 and law of Laplace, 74
 left, 22-26, 37, 39-40, 41, 149, 150, 154-155

Ventricle, left (Cont'd)

- and aging, 133, 137
- in exercise, 133-139
- hypertrophy, 372
- respiratory quotient, 50, 51
- stroke volume, 21-25, 11, 133, 137, 138, 216
- in mitral stenosis, 78
- premature beats, 128-129, 145
- pressure, and blood volume, 56
- right, 22, 50, 81
- septal defect, 251
- tachycardia, 173

118 156

- 341
- West Pennsylvania Heart Association, 341
- West Virginia, 435
- Wisconsin, 435, 441
- Wolff-Parkinson-White syndrome, 252, 371
- Work capacity classification, 125-127, 140-149
 - 305-413, 434
 - and age, 308, 323-324, 331, 344
 - at Altro workshops, 349-350
 - and aneurysm, 123, 319-321
 - and angina, 345, 372
 - and arrhythmias, 333, 371, 372
 - and arterial oxygen content, 93
 - and atherosclerotic heart disease, 123, 127, 326, 329, 331-332, 334, 345, 372, 386
 - change after illness, 312, 334, 509
 - in congenital heart disease, 123, 326-329, 331-332, 345, 386
 - cost of operation, 346
 - criteria, 343, 502-506
 - and educational background, 334
 - Esso Baton Rouge Refinery, 368-374
 - examination procedures, 344
 - electrocardiography, 128-131, 319-321, 386
 - exercise tolerance tests, 125, 128-129, 143, 145-146, 159, 333, 403
 - and heart function, 140-147, 312, 331-332, 334, 352-354, 357, 369, 378, 505, 509
 - and therapeutic aids, 319-321
 - and heart size, 320, 321, 372
 - and hospitalization, 344
 - in hypertensive disease, 331-332, 345
 - and job effectiveness, 373-374
 - in myocardium infarction, 123, 127, 419-321, 333, 345
 - and obesity, 320
 - and severity of disease, 403-409
 - social problems in, 518
 - therapeutic, 319-321, 331-332
 - units approach, 305, 331-332, 340-347, 411-412
 - Bellevue Hospital, 305-310, 420, 424
 - in Boston, 311-321
 - in Cleveland, 330-338, 411-412, 420
 - educational possibilities, 412
 - and industry, 413
 - in Philadelphia, 322-329, 341
 - and referral status, 347
 - in United Kingdom, 400-409
 - Women's Compensation System, 417-513
 - accident and causal relationship, 463, 480, 509
 - administration of law, 451-452, 512
 - and concept, 461
 - guideposts for, 481, 512
 - by layman, 468-469, 480, 510
 - medical guidance and opinions, 468, 480-481

awards, 507

- compensable period, 450-451
- for disfigurement, 150
- lump sum basis, 450
- in New York State, 422
- for several injuries, 469
- claims, 426, 450, 463-465
- casual relationships, 386, 388, 393, 394, 405, 426-431, 467, 488, 491, 508, 509
- criteria for, 461-462, 510
- disability, 394, 418-419, 457
- and job security, 513
- liability measurement, 388, 493, 394, 422, 428, 450, 457, 460-466, 510
- for sickness in garment workers, 388
- types, 427-428
- unusual exertion theory, 482
- collection of information for, 470-472, 475, 477, 510
- costs, 442-443, 448, 450, 453, 457
- court procedures, 478-479, 510
- curative function, 443-444
- definitions, 438, 462-463
- disadvantages of current laws, 509-510
- and Utah, 393, 487-498
- education of young cardiac, 419
- employment, 422
 - and development of cardiac pathology, 426-431, 480-481, 512
 - and penetrating wounds, 427, 494
- expert opinion, 464-465, 468-469, 478, 510
- and burden of proof, 511
- and court-appointed experts, 476
- facts for, 476, 491
- medical, 471-472, 477, 489, 490-491, 510
- medical and legal, 469, 477, 510
- mobilization, 472-475, 510
- Occupational Disease Panel, 482-483
- of partisan experts, 474, 477
- qualifications or need, 473-474, 510
- susceptibility, 481
- and tailored testimony, 482
- "extra coverage" for added premiums, 458
- features of compensation, 447
- historical development, 439-441, 508
- litigation, 344, 467, 471-472, 481, 507
- medical witnesses, 467-479, 492-494, 510-511
- merit rating, 457
- Model Code of Evidence, 476
- Model Expert Testimony Act, 476
- and nonoccupational diseases, 457
- persons presently covered, 451
- phases, 441
 - compensation, 446-453
 - prevention, 442-443
- and pre-existing defect, 423, 428-431, 481, 492-493, 495, 508
- rehabilitation, 441, 454-459, 507-513
- report of injury to employer, 470-471
- responsibility for effects of injury, 422, 423, 426, 439-441, 462, 466, 510
- and second injury laws, 435, 508
- services evaluation, 454-459
- as social insurance, 455
- specialization of tribunals, 475
- state agencies, 458
- Sub-Committee of American Heart Association, 422
- and time for manifestation of symptoms, 481
- in United Kingdom, 409, 420
- Wyoming, heart injury laws, 435